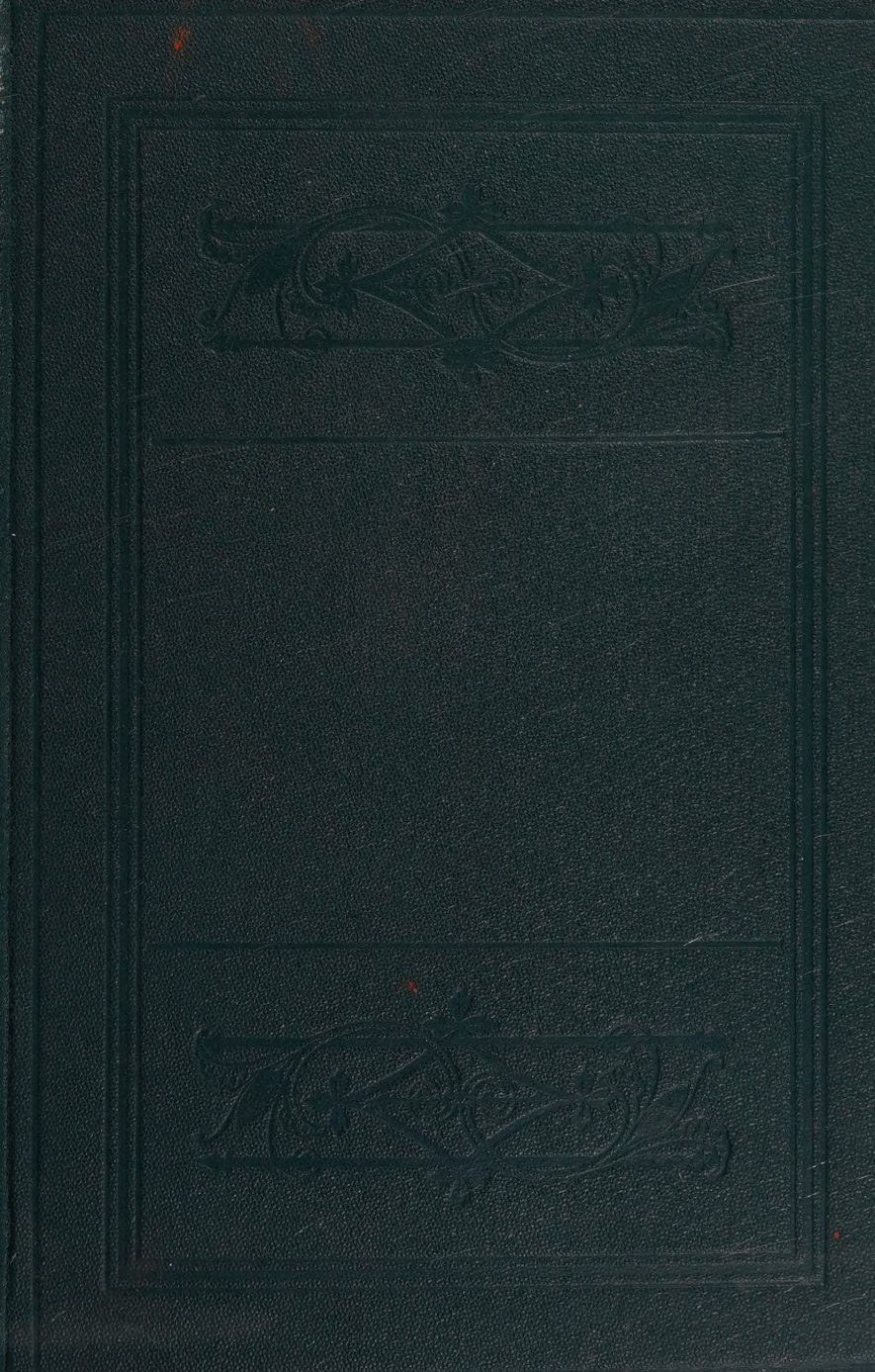

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SURGICAL PATHOLOGY

AND

MORBID ANATOMY

SURGICAL PATHOLOGY

AND

MORBID ANATOMY

BY

ANTHONY A. BOWLBY, F.R.C.S.

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SURGEON TO THE ALEXANDRA HOSPITAL FOR HIP DISEASE

THIRD EDITION

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TO

THE STUDENTS

OF

ST. BARTHOLOMEW'S HOSPITAL

THIS WORK IS

Dedicated

BY THEIR SINCERE FRIEND

THE AUTHOR

PREFACE TO THIRD EDITION.

The increasing importance of Bacteriology has been recognised in the present edition by contributions from my friend, Dr. Kanthack, the present Lecturer on Pathology at St. Bartholomew's Hospital. He has kindly rewritten for me Chapter III., on "Micro-organisms in their Relation to Pathological Processes," and has added to various chapters, especially in the earlier part of the volume, such information on the various pathogenic bacteria as the scope of the work seemed to demand. I have, in addition, added several chapters and paragraphs on fresh subjects which have of recent years become of increasing interest to surgeons, and have revised the book throughout.

I am again indebted to Mr. Mark for twenty additional drawings, which illustrate chiefly the recently added material.

24 Manchester Square,
April 26, 1895.

PREFACE TO SECOND EDITION.

I HAVE taken the opportunity afforded by the publication of a Second Edition to add several fresh chapters and paragraphs on subjects which were not included in the pages of the former edition.

The chief of the new subjects are Glanders, Actinomycosis and Leprosy, Diseases and Injuries of the Larynx, Cachexia Strumipriva, Acromegaly, and Diseases and Injuries of the Face and Skull. Many additions have been made to other chapters, and the whole book has been carefully revised.

I am indebted to Mr. Mark for twenty-three additional Illustrations, the drawings for which have been made from specimens recently added to the museum of St. Bartholomew's Hospital.

To Mr. James Berry I have again to express my thanks for much valuable assistance in revising the proof-sheets.

43 QUEEN ANNE STREET,
Jan. 12, 1890.

PREFACE TO FIRST EDITION.

My object in writing the following pages has been to describe as simply as possible the various Pathological processes which are of interest to the student of surgery.

With this intention, I have, throughout this book, dealt first with diseases as they are met with during life, and have then described their natural courses and terminations, and the morbid appearances presented by the structures involved.

I have endeavoured, as far as possible, to express the current views of pathologists, especially those of the English school; but have not hesitated to make use of my own observations and of the abundant pathological material supplied by the museum and post-mortem room of St. Bartholomew's Hospital. The space at my disposal and the nature of the work have not permitted many references to the authors consulted, and the few that are made relate chiefly to papers whose contents are not yet generally included in the textbooks.

The drawings with which the book is illustrated are original, and have been made by Mr. T. Godart from specimens now in the museum of St. Bartholomew's Hospital, with the exception of five which are from

specimens in that of St. Thomas's Hospital. I believe that all of them are faithful copies of the morbid appearances they represent. The blocks for printing have been prepared by the "Typo-Etching Company."

My best thanks are due to my friends Mr. James Berry, Mr. D'Arcy Power, and Mr. Edgar Willett for much valuable advice, and for assistance in revising the proof-sheets.

43 Queen Anne Street,
Sept. 1887.

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SURGICAL PATHOLOGY.

CHAPTER I.

HYPERTROPHY AND ATROPHY.

HYPERTROPHY.

Hypertrophy means increase in size, and is a term usually limited to such enlargements as are evidently abnormal in their extent.

The increase in size of any tissue may be the result of an increase in either the number or the size of its constituent elements, the former variety being called numerical, the latter simple; they are frequently coincident. In **true** hypertrophy the various constituent parts of the organ or tissue affected are all equally overgrown; in **false** hypertrophy, although the organ or tissue is larger than natural, the overgrowth is due to an increase of one or more of its constituent elements, to the exclusion of the other. The term hypertrophy is indeed misapplied in such instances, for if it be employed to imply increase in size, it is evident that it should not be used where there is, in addition, alteration in structure.

There are two chief causes of hypertrophy: first, **increased use;** second, **increased blood-supply.** With regard to the former, it may be said that increase of function is always accompanied by increase in growth, so long as the work required be not excessive. True physiological

hypertrophies, the result of increased use, are best seen in the muscles of a healthy person when called upon for greater exertions than are usual, or in the uterus during pregnancy. From a pathological point of view they are most often the result of disease of some other tissue or organ, whose impaired functions are supplemented by increased functional activity in the healthy parts. Examples of such **secondary** or compensatory hypertrophies are common, and good instances are supplied in the hypertrophy of one kidney when the other is diseased, the increase in size of the fibula when the tibia is congenitally absent, and the thickening of the muscular walls of the heart in cases of valvular disease, or of those of the bladder in obstructions caused by enlarged prostate or stricture of the urethra.

The hypertrophy which results from increased blood-supply may occur in tissues which are otherwise normal, but is most often seen in connection with irritative or inflammatory lesions. Hunter's experiment of transplanting the spur of a cock on to its comb affords an excellent example of the former, for the spur, nourished by a greatly increased vascular supply, grew to many times its original size. In the same way, the hair in the neighbourhood of a chronic ulcer or an inflamed joint, though itself healthy, often grows longer and becomes more thick on account of the vascularity of the skin in the neighbourhood of the inflamed area.

More often, however, the overgrowth which is dependent on increased blood-supply is the result of some abnormal condition of the tissue itself. Thus, the irritation of a badly-fitting boot, or the intermittent pressure of a tool on the skin of the palm, often results in the thickening of both the derma and epidermis, and the effects of intermittent pressure on a bone is similarly shown in the formation of new bone from the periosteum. Hypertrophies such as these are called **irritative.** It is worth while to mention yet one other instance, for the results are sometimes very striking. In cases of chronic inflammation kept up by any

cause in the neighbourhood of the epiphysis of a growing bone, the increased supply of blood to the developing tissue is followed by a corresponding increase in growth, and thus the limb on the diseased side may become longer than its fellow. In the tibia such overgrowth is accompanied by curvature, for, being fixed to the fibula, it is unable to grow in length more rapidly than the latter bone, unless at the same time it yields to the resistance offered by the fibula.

In some cases parts become hypertrophied without any apparent cause, and a toe or a finger may continue to grow after the growth of its fellows has ceased, till it becomes more than twice the normal size. In other cases whole limbs become hypertrophied, but we know no more what is the cause of this overgrowth than we know why the growth of any one part of the body ceases when its natural development has been attained.

ATROPHY.

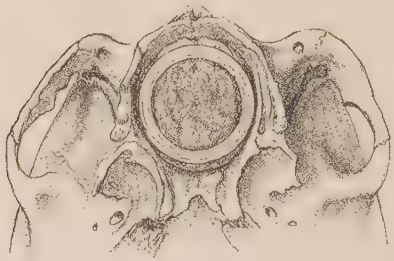
Atrophy means diminution in size without alteration in structure, and, if the term be strictly used, pure atrophies will be found to be of rare occurrence.

Causes.—The most important causes of atrophy are, first, **deficient blood-supply;** second, **deficient use.** The deficient blood-supply is frequently itself the result of some morbid process, and is often caused by pressure. As already mentioned, intermittent pressure, by causing irritation, induces hypertrophy; but **continuous pressure,** on the other hand, induces atrophy. No better instance of this can be desired than the atrophy of all the tissues which is often caused by the pressure of an aneurysm, for not only are the soft structures destroyed, but bones and cartilages may be completely removed. Thus, in the case of an aortic aneurysm, the ribs or the sternum may be either simply eroded or entirely absorbed in their whole thickness, and the bodies of the vertebræ may be similarly affected; this absorption or atrophy results from the obliteration of the blood-vessels by the pressure of the aneurysmal sac.

The specimen depicted in Fig. 1 shows the complete absorption of the whole of the hard palate caused by the constant wearing of a cork inserted to plug an aperture which communicated with the nostrils. The patient was an old woman, and as the hole constantly grew larger, it became necessary to increase the size of the cork, and finally to add to its circumference by wrapping it with strips of tape.

The atrophy which results from deficient use or diminished functional activity is well seen in the condition of a limb in which one of the joints has long been diseased. In such,

FIG. 1.



Part of the Base of a Skull, showing a cork surrounded by several layers of tape, plugging an aperture in the hard palate.

the soft tissues as a whole are diminished in bulk, and muscles, vessels, and nerves all share in the general atrophy. The bones do not escape; they become hollowed out from within, and reduced to mere shells—eccentric atrophy—whilst at the same time their transverse diameter also lessens—concentric atrophy—and thus both eccentric and concentric atrophy go hand in hand. Similar wasting may be seen in the tissues that form an amputation stump, the bone in which is often atrophied to such an extent that it measures less than a third of its normal circumference (Fig. 2). The diminution of the optic nerve after removal of the eyeball, and of the renal artery when the corre-

sponding kidney has been destroyed, afford other good examples of the same process.

Many more varieties of atrophy might be mentioned, but not only are they more suitably discussed in works on general pathology—they are also for the most part physiological rather than pathological. Thus, the atrophy of the thymus gland in childhood and of the breasts after the cessation of menstruation cannot justly be considered as in any way abnormal, and therefore find no place in works on pathology, except for purposes of comparison or illustration. Another cause of atrophy which is commonly mentioned is excessive use, but it is questionable whether this ever induces an atrophy which can be considered pathological.

The other forms of atrophy which are accompanied by fatty changes in the affected tissues, and which are more common than the pure atrophies, will be considered in the ensuing chapter on "Degeneration."

FIG. 2.

The Upper Half of a Humerus from a Stump. The bone is both diminished in its circumference and hollowed out from within, the compact tissue being greatly diminished in thickness.

CHAPTER II.

DEGENERATION.

The term **degeneration** is used to imply an alteration in structure whereby the organ or tissue affected is rendered less able to perform its normal functions, the tissue elements themselves being replaced by material of inferior quality.

FATTY DEGENERATION.

The most common form of degeneration is that named **fatty.** In it the cells and fibres are replaced by fatty granules, and the whole texture of an organ may by a continuance of this process be transformed into fatty matter. Whatever may be the special cause at work in any individual case, all fatty degeneration is caused by a **deficiency in the supply of arterial blood**, and this may be induced in various ways. Thus, an organ by becoming hypertrophied may outgrow its blood-supply—a condition which is well exemplified in cases of heart disease and of obstruction to the outflow of urine. The heart in one case, the bladder in the other, become hypertrophied to meet the increased work thrown upon them, and so long as the vascular supply increases in just proportion all goes well. But the day comes when the tissue outgrows its blood-supply, and then fatty degeneration ensues, the hypertrophied organ becoming dilated, and soon failing to expel its contents. It will thus be seen that hypertrophy and fatty degeneration may co-exist in the same organ. And what is true of hypertrophy applies also to new growths. Many tumours outgrow their vascular supply, and consequently

degenerate in their oldest and most central parts. Thus, in scirrhous carcinomata it is common to find fatty degeneration of the epithelial cells, and in sarcomatous growths many of the cells become filled with granules of oily material. So also with inflammatory products. In all inflammations there is a tendency to fatty change in the exuded leucocytes, and the more chronic the inflammation and the more unhealthy the patient the more noticeable is this condition. It is always best marked in strumous or tubercular inflammation, and is well exemplified by the fatty mass of disintegrated cells which fills a caseous lymphatic gland, or the pulpy synovial membrane seen in strumous joints. Again, in inflammations of arteries, whose intima, it must be remembered, is nourished by osmosis, the exudation cells are peculiarly liable to degenerate and form the so-called atheromatous abscesses and ulcers met with in chronic arteritis. Thus, whether the increase in size in a tissue be due to hypertrophy, to new growth, or to inflammatory exudation, unless the blood-supply increase in proportion to the growth, fatty degeneration will ensue.

But whilst, on the one hand, fatty degeneration may be caused by overgrowth of the tissue to be nourished, on the other it may result from cutting off of the normal blood-supply. Here, again, the heart affords an excellent example, for, when its coronary arteries are atheromatous and its muscle imperfectly supplied with blood, fatty degeneration of the walls of the ventricles commonly results, and may lead to the death of the patient either by failure of the heart's action or by rupture of the ventricle itself. In the brain also, the result of disease of the vessels is fatty degeneration and so-called "softening," a condition which is commonly met with in old people. Considering that atrophy, as we have already seen, is due to the same cause as fatty degeneration, namely, deficient arterial supply, we should rather expect to meet the two processes occurring together; and such indeed is the case. The atrophy of the

tissues in old age is not a simple atrophy, but is almost always accompanied by fatty degeneration, and both are probably to a great extent dependent upon the atheroma of the vessels which is common in old people. Again, in the atrophy which results from disuse there is frequently also fatty change, and the wasting of the glandular tissues in old age, and of the muscles as well as of the bones in useless limbs, is almost always accompanied by degeneration.

If a tissue in a state of fatty degeneration be examined microscopically, its cells and fibres will be seen infiltrated with fatty granules and nuclei, and, in cases of long standing, plates of cholesterine and crystals of stearic acid may be found. If sections are stained in osmic acid, the fatty particles are turned black; whilst if they are exposed to the action of ether, they are rapidly dissolved.

MUCOID DEGENERATION.

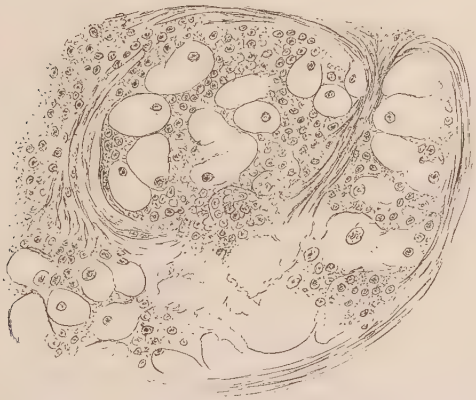
Mucoid degeneration, occurring as a pathological process, has its physiological type in the formation of mucus by the glands of the mucous membrane. In these the protoplasm of the cells is constantly undergoing metamorphosis into a semi-transparent substance, which distends the cell to bursting, and finally escapes from within it. The epithelia of diseased tissue or of new growths may secrete a like material, and in other cases the ground substance between the cells undergoes similar degenerative change. Thus, in the sarcomata, chondromata, and myxomata, it is common to find portions of the new growth quite soft and gelatinous, and a microscopical examination shows the cell-processes interlacing with each other in a mass of homogeneous, mucoid matter which represents the degenerate matrix.

COLLOID DEGENERATION.

Colloid degeneration is closely allied to mucoid, the colloid substance being distinguishable only by the fact that, unlike mucus, it is not coagulated by the addition of acetic acid, and is not rendered opaque by the addition of alcohol.

Colloid degeneration finds its physiological type in the formation of colloid matter by the thyroid gland, where it is produced by the epithelial cells in the same way as is mucus in the mucous glands. Colloid material is of rare occurrence as a pathological product, but is met with in the cells of carcinomata of the stomach, mesentery, and intestines, as well as in new growths of the thyroid and ovary,

FIG. 3.



Section of a Carcinoma of the Breast. Many of the cells are distended with colloid matter, and in the lower part of the section is an irregular cavity caused by the bursting of some such cells.

and more rarely in cancers of the breast (Fig. 3). Entangled in and filling the alveoli of a carcinoma, it gives the appearance of a honeycomb filled with semi-translucent gelatinous matter, and is not easily mistaken for anything else. The occurrence of mucoid or colloid degeneration in new growths in no way affects their malignancy.

CALCAREOUS DEGENERATION.

Calcareous degeneration is a process by which a tissue becomes impregnated with various salts, the most common of which are phosphate and carbonate of lime, together

with a small quantity of magnesium compounds. The deposit of such material in a growing bone is a normal process, but the cause of calcareous degeneration is not clearly understood. It is, however, a well-ascertained fact that impaired nutrition, or even death, of a tissue always precedes calcification, and it is supposed by some authors that special forms of albumin are produced which possess special affinities for lime salts. Calcareous degeneration may occur in any part of the body, but is especially liable to affect the arteries in aged people, as well as the cartilages of the ribs, larynx, and trachea. It is also of common occurrence in the products of previous pathological processes. Thus, fatty and cartilaginous tumours frequently calcify in parts, as do also some sarcomata and fibrous tumours. The products of past inflammations are favourite sites of calcification, and good examples are supplied by caseous lymphatic glands and the cell exudation of chronic arteritis. The clots in blood-vessels also are liable, after the lapse of many years, to become calcified, and form in the veins the so-called vein-stones or phlebolithes.

Microscopic examination of a tissue in an early stage of calcareous degeneration shows small shining granules deposited in the cells and matrix—where such can be distinguished—and occasionally arranged in concentric layers forming "chalky concretions." The addition of a drop or two of hydrochloric acid causes a rapid disappearance of the calcareous matter.

AMYLOID, ALBUMINOID, OR LARDACEOUS DEGENERATION.

In this form of degeneration the tissues are impregnated with an albuminous material which in part takes the place of the diseased structure, and in part is added to it as an infiltration. The organs most commonly affected are the liver, spleen, kidneys, and intestines. The solid viscera are, in advanced cases, much increased in size, and are unduly firm to the touch. On section, they are paler

than natural, homogeneous, and waxy. When portions only of the organ are implicated, the amyloid patches appear as rounded semi-translucent spherules, looking like grains of boiled sago. This appearance is most marked in the spleen, and has given rise to the term of "sago spleen." If iodine be poured over the surface of a diseased organ, the amyloid material is stained a dark red-brown or mahogany colour.

Microscopical examination shows that the amyloid substance is first deposited in the walls of the small blood-vessels, especially in their sub-endothelial cellular tissue. This readily accounts for the patchy distribution of the substance in the affected viscera, the Malpighian bodies in the kidneys and spleen being the seat of the earliest deposit, whilst the arteries which run at right angles to the long axis of the intestine, and the blood-vessels which run parallel to the renal tubules, appear as dark-brown streaks when the part is stained with iodine. The amyloid substance is not stained by logwood or carmine, and in sections prepared with these reagents appears as a homogeneous mass hiding the normal structure. In sections stained with methyl-violet, however, the amyloid matter takes on a bright ruby-red tint, whilst the healthy tissues are stained a deep blue.

The effects of amyloid disease of the viscera are very serious, for, on account of the early implication of the blood-vessels, the nutrition of the part is much interfered with, and, later on, as the glandular elements become affected, the functions of the organ are further impaired. Advanced amyloid disease is thus frequently fatal.

By far the commonest **cause** of amyloid disease is **chronic suppuration** ; it is, however, also met with in syphilis, both congenital and acquired. Cases of strumous caries and of diseased joints are very frequently fatal from this cause, but the affection is not commonly seen until the suppuration has been in progress for some months, nor unless the discharge of pus is considerable. If the cause of

the suppuration be removed—*e.g.*, if the affected limb be amputated before the disease has progressed too far, it is quite possible for recovery to result, and for the diseased viscera to gradually resume their natural size and functions. Thus, albumen may disappear from the urine, and an enlarged liver may gradually recede under the cover of the ribs after an amputation performed for long-continued disease of a joint.

The exact manner in which suppuration causes the deposit of amyloid material is not known, but various theories have been propounded. The best known of these is that of Dr. Dickinson, who has suggested that the drain on the salts of the blood caused by the long-continued secretion of pus results in the deposit of de-alkalised fibrin. It must, however, be remembered that this explanation does not hold good for those cases in which the disease occurs in the subjects of congenital syphilis, and in the absence of all suppuration.

CHAPTER III.

MICRO-ORGANISMS IN THEIR RELATION TO PATHOLOGICAL PROCESSES.

Many important lesions or morbid processes in the body are due to micro-organisms, minute vegetable or animal organisms, most of which are visible only by the aid of the highest powers of the microscope. Here we are only concerned with two great groups, namely, the **schizomycetes** and the **fungi**, both of which belong to the vegetable kingdom. Recently, the animal organisms have obtained a certain prominence on account of researches made on the nature of cancerous processes. But these are so much obscured by doubt and incomplete observation that a few words will suffice for their consideration (*v.* Chapter XX.)

The **schizomycetes** are extremely minute organisms, consisting of protoplasm which, as a rule, presents no definite structure, but generally stains readily with simple aniline dyes. Chemical analysis shows that these organisms contain Hydrogen, Nitrogen, Oxygen, Carbon, Phosphorus, Sulphur, Magnesium, Calcium, and Potassium. They all multiply by fission, but some in addition reproduce themselves by spore-formation.

The schizomycetes are subdivided, according to their shape and size, into the following classes :—(*a*) Micrococci, (*b*) bacilli, (*c*) vibrios, and (*d*) spirilla.

Micrococci are round or oval, lanceolate organisms, as a rule as broad as they are long. They may occur in pairs (diplococci) or in groups of fours (tetracocci); in short and long

chains (streptococci), or in masses forming a "zoogloea," which consists of numbers of micrococci held together by

FIG. 4.

(*a*) Staphylococci.

(*b*) Streptococci.

(*c*) Diplococci (Pneumococcus).

(*d*) Anthrax Bacilli.

(*e*) Anthrax Bacilli with Spores.

(*f*) Vibrios and Spirillum.

TYPES OF MICRO-ORGANISMS : FROM FLÜGGE'S "HYGIENE."

some mucilaginous material. Examples of these various forms are—the diplococcus pneumoniæ, the micrococcus

tetragonus, the streptococcus pyogenes, and the staphylococcus pyogenes aureus.

The diplococci and tetracocci are often surrounded by a distinct capsule; they change, however, not infrequently in their type when grown in artificial media, the former developing into streptococci and the latter into staphylococci.

Bacilli are rod-shaped organisms with rounded or pointed ends, whose length exceeds their breadth; they multiply by fission, but many of them also by spore-formation. The *spores* are highly refractive and resistent, oval or round bodies, which stain with difficulty. They occupy either the centre of the bacillus, as in the case of the bacillus anthracis, or they are terminal, giving the bacillus the appearance of a drumstick (*e.g.*, tetanus bacillus). Spores are developed under special conditions; thus, the anthrax bacillus never forms spores in the animal body; it requires oxygen, and a favourable temperature and soil. A temperature of 42·5° C. completely prevents the spore-formation; and so also does the addition of a minute trace of carbolic acid to the artificial medium in which the bacillus is grown. A bacillus never possesses more than one spore.

Spirilla are organisms of a spiral or corkscrew shape; twisted in their longitudinal axis. They vary much in length, and, with the exception of the vibrios, are of little importance, the spirillum of recurrent fever being the only known pathogenic typical spirillum met with.

The *vibrios* probably belong to the group of spirilla. They were once regarded as bacilli which are curved instead of being straight, but since they often grow into typical short or long spirals, they are better included in this class. The most familiar example of a vibrio is the organism of Asiatic cholera, which, on account of its resemblance to a comma, was called by Koch the "comma bacillus."

Many of the bacilli, spirilla and vibrios are motile—*i.e.*, they possess the power of spontaneous movement. Such organisms are generally found to have cilia or flagella. Some

bacilli, like the typhoid bacillus, bristle with flagella, while others have only one or two cilia. Many vibrios, for instance, possess but a single terminal flagellum. These processes can be seen only with special staining methods.

Fungi are much more complex and highly developed than the schizomycetes ; they are distinguished by their mycelium, a meshwork of filaments, which forms the essential part of the vegetating organism. Surgical pathology is concerned which only a few fungi, of which the most important are the Aspergillus niger, which is sometimes found in cases of chronic suppuration (otorrhœa), the Actinomyces, and its allied forms of Streptothrix, which will be described in Chapter XIII.

A few words must be said on the *pleomorphism* of many organisms. Apparently the connection between the schizomycetes and fungi is closer than one would suspect at first sight. Under special conditions, for instance, some bacilli form an atypical mycelium (*e.g.*, bacilli of diphtheria), and some fungi, like the actinomyces, when artificially grown, pass through a bacillary phase. Vibrios especially are highly pleomorphic, developing at times as typical spirilla or again as ordinary bacilli.

Involution must be carefully distinguished from pleomorphism. By involution forms we understand the various changes in shape or size which micro-organisms undergo with age or want of nutrition. These are only temporary, and as soon as the micro-organism is again placed in favourable conditions it will resume its original characters.

CONDITIONS OF LIFE.

We are comparatively ignorant of the actual or real conditions under which the fungi and schizomycetes live in nature. It is well known that micro-organisms cannot thrive beyond a certain altitude or beyond a certain depth of soil; again, that there is a limit to the heat and cold which they are able to bear. These limits are very variable: organisms are found in ice and also in hot springs.

Studying microbic life in the laboratory, it is found that these minute vegetable beings require food, moisture, and warmth.

The **quality of the food or soil** varies considerably. For artificial cultivation the following nutrient media are generally used: Potatoes, beef-broth, beef-peptone-gelatine, and beef-peptone-agar-agar, or blood serum in a state of coagulation. But of much greater importance is the question of parasitism—*i.e.*, whether an organism is capable of growth in living animal tissues, or capable only of growth in dead, dying, or decomposed tissues—whether it is capable of thriving in the animal body or not. In the former case we have to deal with **parasites**, in the latter with **saprophytes.** Now, many microbes are able to flourish in the animal body as well as outside. Accordingly they may be classified in the following manner:

1. Obligatory parasites: organisms capable only of thriving in the animal body.
2. Facultative saprophytes: parasitic organisms, which under special conditions may grow outside the animal body as saprophytes.
3. Facultative parasites: these are preferably saprophytic, but under special conditions become parasitic.
4. Obligatory saprophytes: organisms incapable of thriving in the animal body.

This classification is of great importance in the consideration of the ætiology of microbic diseases. Affections due to obligatory parasites – *e.g.*, gonorrhœa, syphilis, rabies – can be acquired only from a being already affected; while diseases due either to facultative parasites or to saprophytes have a far wider field of infection. Tuberculosis, for instance, which is caused by the bacillus of tuberculosis, a facultative saprophyte, may be acquired directly from an individual already attacked, but the bacillus may gain access to the body also through food, inhaled dust, &c. As far as surgical pathology is concerned, we may arrange the most important schizomycetes, and the diseases produced by them, in the following way:

(*a*) Obligatory parasites: Gonorrhœa
Syphilis
Rabies.

(*b*) Facultative saprophytes: Tuberculosis
Glanders
Diphtheria
Erysipelas
Traumatic infections.

(*c*) Facultative parasites: Anthrax
Tetanus
Malignant œdema.

Aërobic and Anaërobic Organisms.—Oxygen is essential for the life of many of the schizomycetes. There is, however, a considerable number of organisms which can thrive only in an atmosphere devoid of oxygen. The latter are called "anaërobic," the former "aërobic." Many organisms which generally grow under aërobic conditions often also will grow anaërobically. Accordingly, we distinguish:

(1) Obligatory anaërobic organisms—*i.e.*, organisms which require an atmosphere free from oxygen.

(2) Facultative anaërobic organisms—*i.e.*, organisms which under ordinary conditions are aërobic, but which are readily accustomed to anaërobiosis.

(3) Obligatory aërobic organisms—*i.e.*, organisms which cannot exist without oxygen.

In surgical pathology we meet with two important obligatory anaërobic bacilli—viz., the bacillus of tetanus and the bacillus of malignant œdema. Many pathogenic varieties are facultative anaërobic organisms, and this is a point of some importance, because it is evident that in the tissues of the body the supply of free oxygen must be very limited. Thus, all the pyogenic germs are readily trained to an anaërobic life.

Water or moisture is necessary for the growth of all micro-organisms. All living animal tissues contain in themselves a sufficiency. Many forms of organisms are killed

by drying; those, however, which are capable of spore-formation readily survive drying, and so also do others, like the tubercle bacillus and the pyogenic cocci, which do not multiply by means of spores. This resistance is important from the point of view of antisepsis and disinfection; it is necessary to apply the disinfectant not only to the wound and the body, but also to procure an asepsis beyond the limits of the body.

Most micro-organisms prefer an **alkaline** soil for their growth, such as they find in most tissues. There are, however, some which thrive well on acid media. Thus, in acid urine organisms are readily found, and the gonococcus, as a matter of fact is said to prefer acidity; in acid saliva also, innumerable bacilli, cocci, and vibrios are found. Nevertheless, generally speaking, a faintly alkaline or a neutral soil is best for microbic life.

Rest.—Almost all schizomycetes and fungi grow best when at rest, and many of them can be prevented from growing to the fullest extent of virulence or from multiplying or from sporulation by keeping the fluid in which they are suspended in a state of constant motion.

Light.—Sunlight and even diffuse daylight are detrimental to the growth and development of many pathogenic organisms. Thus, the direct rays of the sun—even of the winter sun—will destroy readily anthrax bacilli and their spores, tubercle bacilli and other organisms. This is not due to the heat of the sun; and it has been shown that the blue rays of the spectrum are far more bactericidal than red rays. Diffuse light will readily diminish the virulence of tubercle bacilli. Light, therefore, is an important element in the treatment of many forms of infective disease, though too much must not be expected from it as a practical antiseptic. Most, if not all, the pathogenic germs grow best in the dark.

Temperature.—The pathogenic micro-organisms mostly thrive best at the temperature of the animal body, but, as already mentioned, the limits of the temperature favourable

or unfavourable for their life vary greatly, some being capable of surviving great alterations. Thus, the tubercle bacillus will withstand heating for several minutes to 60° C., which will destroy the bacillus of typhoid fever or the vibrio of cholera without fail. Spores can withstand even high temperatures; but most organisms are destroyed after being heated to 100° C. for several minutes.

Pathogenic organisms have been mentioned repeatedly without a definition having been supplied. It is difficult to give a definition which covers all the properties of the pathogenic organisms, or includes all the members of this class. Some organisms when injected or introduced into the animal body produce a certain lesion, or a group of symptoms which will not appear without the entrance of such organisms into the body. These are called pathogenic germs. They are characterised by their power of multiplying in, and acting upon, the living tissues—*i.e.*, they are either facultative or obligatory parasites or facultative saprophytes. They manufacture chemical products, which when absorbed cause constitutional symptoms, or act on the tissues, producing certain lesions, destructive or formative. Sometimes a distinctive lesion appears, which is characteristic and *sui generis*, and is accompanied by equally characteristic symptoms. In such cases we speak of **specific** organisms and **specific** lesions or diseases. It must be remembered, however, that different species of bacilli and cocci may and do produce identical lesions and symptoms. Suppuration, for instance, is not strictly a specific lesion, because it is not confined to a single organism, but its appearance can be traced to a number of different pyogenic cocci and bacilli. It has already been mentioned that some of the pathogenic organisms are capable of acting on healthy tissues only, namely, the obligatory parasites. Many, if not most, of them, however, find a suitable habitat only in the tissues which have been injured, whose vital powers are lowered by inflammation, injury, or other pathological conditions, as for instance, general ill-health, which predisposes the body

to an attack by the organisms. These are the facultative saprophytes and facultative parasites. There are, however, also conditions, when strict or obligatory saprophytes will lead to disease, as, *e.g.*, when organisms of decomposition find a soil on necrosed or dead tissues. Here they will multiply and form their poisons, which are absorbed, causing the constitutional symptoms of septic intoxication. In most lesions several organisms occur together, so that there is a multiple infection, as, *e.g.*, in cases of tetanus, where pyogenic organisms are found in company with the bacillus of tetanus, or in cases of septicæmia, where saprophytic organisms assist the pyogenic cocci. In such cases one organism may further and help another by a process of "symbiosis," thereby leading to a serious exacerbation of the lesions and symptoms.

Before an organism can be proclaimed the specific cause of any disease, certain conditions must be fulfilled, viz.:

1. The organism must be found in every instance of the disease, either by means of microscopical or bacterioscopic examination—

2. It must not be found in any other disease—

3. Inoculations of susceptible animals with the organism artificially cultivated must reproduce the original lesion, and in this experimental lesion the same organism must be found, to the exclusion of all others—

4. Transferred from animal to animal, it must invariably produce the same lesion—

5. The poisons or toxines prepared either from artificial cultures or separated out of the diseased body, and injected into a susceptible animal, must lead to symptoms and processes characteristic of, and compatible with, the original disease.

Mode of action of pathogenic organisms.—The pathogenic organisms may produce definite lesions or symptoms—(1) through mechanical interference, and (2) through their chemical products. As far as mechanical interference is concerned, they may obstruct the capillaries and smaller vessels, and thus possibly lead to infective embolism, which

may result in rapid death. Again, as in diphtheria, infective membranes may be found in vital organs, or, as in ulcerative endocarditis, infective vegetations may develop on the flaps of the cardiac valves. Local œdema or necrosis is often no doubt partially due to mechanical vascular lesions, but the more important and characteristic morbid changes are unquestionably due to the absorption of the microbic chemical products, or to their action on the tissues. In every infective process, then, **intoxication** plays a great, if not the greatest, part. This is a point of some importance, because until recently narrow distinctions were drawn between infective and intoxicative processes. By an **infection** we ought to understand the entrance or introduction of pathogenic organisms into the body; but in every infection we must have also an intoxication with the poisons or toxines manufactured by these organisms.

Bacterial Poisons and Toxines.—Our knowledge of the chemical nature of the metabolic products of micro-organisms is still uncertain and incomplete. While we are still unacquainted with the chemistry of the tissues, while proteids and peptones, or albumoses, are little more than names, it is almost useless to speculate on the chemical changes induced by the micro-organisms in the living tissues. In artificial media, such as broth, two processes always take place: (*a*) destructive or assimilative, (*b*) constructive or secretive. The organisms take up certain substances or elements, thereby splitting up complex molecules or atomic groups, and thus altering the original medium in which they were placed; but on the other hand they also secrete complex substances which they have built up from the simpler assimilated bodies.

Some organisms produce acids, others pigments; some form aromatic compounds, others various kinds of gases. Ferments and other complex bodies are also found among their metabolic products. These complex substances require special attention: they include the ptomaïnes (so-called animal alkaloids), the albumoses, the toxalbumines

(peptones or globulines), and the toxines or ferment-like albuminoid bodies. We are still ignorant as to the true nature of the "specific" chemical poisons. Fashion often models scientific opinion. Thus at one time ptomaïnes were considered to be responsible for the specific lesions, but this view was soon shown to be untenable. The albumoses had their turn, and undoubtedly albumoses are formed by many organisms when grown in albuminous nutrient media, as, for instance, by the bacillus of anthrax and of diphtheria. Others, however, form no albumoses, or the albumoses produced by them have no specific toxic power. To a great extent the nature of the chemical substance—*i.e.*, whether it be an albumose, a peptone, or a globuline, or shortly a toxalbumine—seems to depend on the composition of the nutrient medium, and it seems that when the organisms are grown in non-albuminous solutions, as, *e.g.*, in urine or aspartate of sodium solutions, no such toxalbumines are formed, but indefinite albuminoid bodies or toxines. Some organisms, as, *e.g.*, the bacillus of diphtheria, manufacture true unorganised enzymes, while others also act themselves as living ferments. We see, then, that the chemical activity of pathogenic germs is a most complicated process, still shrouded in obscurity; and it must not be forgotten that, as the chemical substances of the microbic metabolism are multiple and complex, so also the symptoms produced by them must be multiple and complex. Thus, at the seat of infection the organisms may act as ferments, splitting off toxic substances from the tissues; at the same time, ptomaïnes, toxalbumines, or toxines, or fermentative products, may be formed, which are absorbed into the circulation. These few remarks must suffice as a short sketch of a doubtful subject.

Susceptibility and Predisposition.—An organism which is pathogenic for a particular animal species is not necessarily pathogenic for another; it may be a closely allied species. The infective power is therefore a relative quantity, which depends on particular, at present more or

less unknown, changes and conditions of the tissues. We cannot tell why the grey mouse so readily succumbs to an inoculation with the tubercle bacillus, while the white mouse resists it as easily. In the one case there is a **natural predisposition** or **susceptibility**; in the other case a **natural resistance** or **immunity**. Similarly, we find that some individuals are highly susceptible to certain infective diseases, while others show a marked resistance. This susceptibility is often **acquired** in some way or another. Experimentally it has been demonstrated that hunger and starvation, fatigue and over-exertion, enforced thirst, and errors of diet break down a natural resistance and produce an acquired predisposition. The same may be achieved by an injection of chemical and bacterial poisons into the animal body. These are considerations of great importance, because they throw some light on the nature and meaning of predisposition which clinically has always been recognised.

As the predisposition may be either natural or acquired, so also the resistance against, or the immunity from, an infective lesion may be (1) natural, and (2) acquired. It may be acquired in various ways, of which the most important ones will be mentioned here. (*a*) Recovery from an infective disease, as is well known, often leads to immunity, temporary or permanent; (*b*) inoculation with the attenuated virus, as, *e.g.*, in the case of anthrax; or (*c*) repeated inoculations with small doses of virulent microbic cultures; or (*d*) small doses of their toxines and poisons will produce the same result. (*e*) In some cases chemical agents, or (*f*) inoculations with antagonistic micro-organisms, have rendered animals insusceptible. (*g*) Lastly, the greatest triumph of recent bacteriological research is the immunisation by means of the injection of serum obtained from immunised animals, a process which has been used on man in diphtheria and tetanus. (*h*) It must be mentioned also that an immunity may be transmitted from the artificially immunised parent to its offspring.

The **causes of immunity** are still under discussion, and no exclusive theory will explain its nature.

(1) The oldest view was the **exhaustion** theory, according to which a specific organism growing in the animal body exhausts the store of nutrient material, so that the organism must perish. The tissues are assumed to remain permanently in this exhausted condition.

(2) This hypothesis made room for the **retention** theory, which implies a storing-up in the tissues of the metabolic products of the specific organism which renders the animal body an unsuitable soil, just as in the case of yeast, the alcohol produced by the fermentation destroys the life of the yeast cell. These products must be supposed to be retained by the tissues for years.

(3) Metchnikoff explained immunity by the **phagocytic** theory; that is, he showed that in an immune animal the bacteria are ingested and digested by wandering and fixed mesoblastic or mesenchymatous cells, which he called phagocytes. This view describes a common and constant phenomenon of immunity, but leaves its cause unexplained.

(4) The **antitoxic** theory is the most recent. We may have either a gradually acquired tolerance towards the toxines, or certain unknown changes are produced in the tissues and tissue juices which are capable of neutralising the microbic toxines and rendering them harmless.

Undoubtedly, as immunisation is a process which may be effected in various ways, and as there is no one general method applicable to all cases, so also immunity must depend on a series of causes, and cannot be explained by any one exclusive theory, however ingenious.

DISTRIBUTION OF SCHIZOMYCETES OUTSIDE AND WITHIN THE BODY.

Although at certain depths and altitudes there is no bacterial or microbic life, yet as far as our ordinary environments are concerned we are everywhere surrounded

by micro-organisms. Wherever there is dust we find schizomycetes; the soil and water also contain them. It is evident that if the surroundings of man are so impregnated with organisms, the latter must find an easy entrance to the body by the respiratory and alimentary tracts or through open wounds. And such indeed is the case. In every act of respiration they pass in numbers into the respiratory passages, and during eating they are carried into the mouth and alimentary canal. Fortunately, however, the larger number of them are unable to find a suitable soil in the healthy and living tissues for setting up morbid lesions and changes. Many of them are destroyed by the cells or die; others are somehow or other impaired in their activity or virulence. Thus, the saliva and bronchial mucus of many persons contain the pneumococcus, the intestines harbour the bacillus coli commune, and it seems that the diphtheria bacillus also is a guest of many normal tonsils. All these organisms are highly pathogenic, but somehow they do not give evidence of their activity except under special conditions. Again, the micrococci of suppuration are found everywhere in the dust and air around us, even in the epidermis and sweat glands; the soil teams with the deadly tetanus bacilli, and the organisms of putrefaction are everywhere ready to attack dead or dying, necrosed or necrosing tissues. The germs, therefore, are present, and it is evident, that given an entrance into the body or tissues, and also suitable conditions for their full development and physiological activity (*i.e.*, the predisposition), morbid or septic changes must follow.

Broadly speaking, it may be said that the micro-organisms reveal their presence by one of three processess, viz.: (1) putrefaction; (2) fermentation; (3) infective disease. Asepsis, Sterilisation, and Disinfection are the means of preventing their activity.

(1) **Decomposition and putrefaction** are the result of saprophytic organisms. Formerly the latter were included under the term bacterium termo, but it is now well

known that under this designation many forms have been included, of which the various kinds of Proteus are the commonest. The putrefactive organisms include both aërobic and anaërobic germs, and are widely distributed outside the body. They have no power over living tissues; thus, intravenous injections of Proteus bacilli have no effect on healthy rabbits. They require dead or devitalised tissues—*e.g.*, a gangrenous limb, the injured surface of the puerperal uterus, or a strangulated knuckle of intestine. Under such conditions they develop rapidly, and produce their toxines and enzymes, which cause intoxication and decomposition.

In the absence of such organisms, dead tissues do not decompose, and may be kept for any length of time in sterilised vessels, in aseptic and antiseptic fluids. Certain degrees of high and low temperature can also prevent or delay putrefaction. The chemical and fermentative products of these saprophytes, which at one time were called ptomaïnes, but of whose real nature we know so little that it is best to speak of them generally as toxines, are extremely irritating to the living tissues, and excite in them an acute inflammation which often terminates in suppuration. The term **septic** has been applied to all such inflammations as result from the presence of decomposing material, whilst the constitutional conditions which arise if the septic products of decomposition are absorbed are well known under the name of "**sapræmia**." It must not be forgotten, however, that all such terms denote familiar clinical conditions, and were adopted at a time when the pathology of these conditions was not properly understood. They must therefore be used with caution and a full appreciation of their inadequacy.

It is evident that these putrefactive organisms are a pathological agent of great importance, and it must be further remembered that their action is not limited to large masses of dead tissue, but that they are equally potent in the smallest drop of fluid, so that serum, blood, or pus may

undergo putrefactive changes when extravasated in or over the tissues, and may thus excite inflammation and suppuration amongst them, whilst if the septic products are absorbed the various constitutional states mentioned above will supervene.

(2) **Fermentation** probably accompanies most putrefactive or infective processes, and has already been discussed above. As yet our knowledge of the nature and action of ferments and enzymes is limited. Numerous good examples of enzymes, the result of cellular activity, are to be found in the human body. Such are pepsin, trypsin, &c., bodies which are capable of causing very definite changes in the chemical composition of the fluids in which they exist. These may be compared with similar enzymes elaborated by the micro-organisms. Thus, all schizomycetes which liquefy gelatine manufacture a proteolytic enzyme, and it seems also that some pathogenic organisms, as, *e.g.*, anthrax and diphtheritic bacilli, have the power of producing enzymes, or of acting as ferments capable of splitting up albumin into toxic albumoses.

(3) The **infective nature** of pathogenic organisms has been considered at length above. Within the past few years many specific diseases have been shown to be the result of the action of one or other of the schizomycetes. Their exact mode of action differs according to the variety of the organism. Some accumulate and multiply in the minute blood-vessels, and diffuse their toxines by means of the circulation. Others cause local acute inflammations, others more chronic changes. But in all cases, it may be mentioned again, the organisms act by producing certain chemical substances which when absorbed act as poisons to the various tissues and lead to severe constitutional symptoms.

CHAPTER IV.

INFLAMMATION.

The best definition of inflammation is that given by Burdon Sanderson, who describes it briefly as "the succession of changes which occurs in a living tissue when it is injured, provided that the injury is not of such a degree as at once to destroy its structure and vitality."

It has been ascertained by experiments on various animals, that the phenomena of inflammation are practically of the same nature wherever inflamed structures are found. It is therefore unnecessary to detail the various experiments alluded to, and we may pass at once to a description of the process itself.

CHANGES IN THE BLOOD-VESSELS AND THE CIRCULATION.

It is in the blood-vessels and their contained blood that the earliest of the changes which occur in inflammation are to be found. The first effect of any injury is a **dilatation** of the vessels—arteries, veins, and capillaries—a condition which is first noticeable and most marked in the arteries. The immediate result of this alteration in the calibre of the vessels is an increased supply of blood to the tissues, and at first an increase in the rapidity of the blood-stream. To this increased afflux the name **determination of blood** has been applied.

After a variable time, the rapidity of the blood-stream diminishes, the retardation sometimes taking place very suddenly, and being first seen in the veins. As an imme-

diate result, the current in the capillaries, and then in the arteries, becomes in turn slower, and pulsation is plainly visible in the smallest arterioles.

If attention is paid to the behaviour of the blood-cells themselves, it will be seen that, during the stage of "determination of blood" the red blood-cells in the veins are swept along in the middle of the stream, in what is named the "axial current." The white cells float on the outer side of the mid-stream near the vessel-wall. When the blood-current becomes slowed, the white cells exhibit a marked tendency to adhere to the walls of the veins, and on account of this stickiness they gradually accumulate immediately inside the vessel. And not only do they tend to adhere to the vein-wall, but they also adhere to one another, and thus the vessel becomes lined by layers of leucocytes, which constantly gather fresh companions from the passing blood.

In this way the lumen of the vessel is narrowed, but at first the axial current sweeps by with unabated velocity. Gradually, however, this velocity is diminished, for the red blood-cells cohere and form rouleaux, and thus pass less readily through the smallest vessels. Finally, complete stoppage, or **stasis,** ensues, and the smallest veins and arterioles are seen to be filled with blood in which no movement takes place. If stasis persists, the nutrition of the vessel-wall is cut off, and it dies, with the result that its contained blood coagulates.

EXUDATION.

In any inflamed area the contents of the blood-vessels exude in greater quantities than in the natural state. It must be remembered that, in the ordinary processes of nutrition of a healthy tissue, the parts around the vessels receive their supplies of new material from, and yield up their waste products to, the white blood-cells and the serum which are constantly traversing them after passing out of the smaller veins and capillaries. But in an inflamed area

there is not only an increase in the amount of the exudation ; there is also an alteration in its composition.

In the earlier stages of inflammation, and in all inflammations of slight intensity, there is merely an increase in the exudation of **serum**, a so-called **serous exudation**, in consequence of which the tissues become more succulent and œdematous, but are otherwise unaltered. The fluid, if drawn off, is found to be almost pure blood-serum, containing very few leucocytes, and no coagulable material. The best example of a simple serous exudation is afforded by a blister. In the early stage of all inflammations, the greater part of the exuded serum is removed from the inflamed area by the lymphatics, and experiments have shown that the flow in the lymphatic channels is thus greatly increased.

As the inflammation progresses and becomes more intense, so the character of the exuded fluid becomes altered. Instead of being watery or serous, it is coagulable and turbid, and to fluid such as this the name of **plastic exudation** is applied. It differs from the serous exudation in that it contains the fibrin-forming elements of the blood as well as numerous leucocytes. If drawn off, it is found to form a firm white clot, and in the tissues also it coagulates. Fluid such as this is often called "**lymph**," and may be seen to perfection in cases of plastic iritis, forming yellowish-white beads or drops in the anterior chamber and on the surface of the iris. On account of its coagulability its flow through the lymphatics is impeded, and after a time it clots in and occludes the lymphatic channels. As an immediate result the tissues which these channels should drain become swollen with retained fluid, and, in consequence of the coagulation of the exudation, they become "indurated" and "brawny" in a manner which is typical of plastic inflammation, and is the common accompaniment of many deeply-seated suppurations.

Diapedesis.—Even in the earlier stages of an acute inflammation, and also in inflammations of but slight

intensity, the white blood-corpuscles escape from the smaller veins. The more advanced the inflammation, and the greater its intensity, within certain limits, the greater are the numbers in which they escape.

If a small vessel be watched, it will be seen that soon after the slackening of the blood-current, and the adhesion of the white blood-cells to the vessel-wall, the contour of the vein is bulged at one or more spots. These localised projections gradually increase, and form rounded or button-like prominences on the outside of the vein. Soon they become further separated from the vessel, and appear to be attached to it by a stalk. Finally, this stalk gives way, and it is now seen that a leucocyte has slowly worked its way through the vein and is free in the surrounding tissue. In this manner varying numbers of white blood-cells escape from the vessel, together with some of the fluid constituents of the blood.

The diapedesis of leucocytes is always first noticed in the veins, and then in the capillaries and the smallest arterioles. In very acute inflammations the red blood-cells as well as the white escape from the vessels, and sometimes in such numbers that the tissue may appear to be infiltrated with blood.

It will thus be seen that in inflammation any or all of the various constituents of the blood may escape. In slight inflammations, and in the earliest stages of the acute forms, only the serum exudes, but as the inflammatory process progresses the fibrin-forming elements, the white blood-cells, and finally the red blood-cells, pass into the surrounding tissues.

In some cases of inflammation diapedesis is absent and no leucocytes appear at the seat of lesion. Recently the term "chemiotaxis" has been applied to the phenomenon of diapedesis or its absence. It is assumed that certain irritating substances attract the leucocytes, while others repel them; so that in the one case we have a chemical attraction or **positive chemiotaxis**, in the other a

chemical repulsion of leucocytes or **negative chemiotaxis.** It is better to adhere to the older terms of diapedesis, or migration of white corpuscles, since they describe merely a phenomenon without attempting to explain its cause.

CHANGES IN THE INFLAMED TISSUES.

There is one change which is common to all inflamed tissues, and that is **softening.** If a piece of connective tissue in a state of inflammation be examined under the microscope, it will be seen that the fibres are swollen, and their outlines are blurred and indistinct. Separating the bundles of fibrous tissue and the individual fibres are numerous leucocytes, which are here and there collected into masses and render all other objects indistinct. It is probable that the softening of the tissues is due in part to absorption by them of the increased fluid exudation in which they are soaked, and also to the destructive or digestive action of the leucocytes, for in many acute inflammations the tissues undergo molecular death. Amidst the swollen and blurred structures, numerous capillaries are seen. Some of these are simply old channels which have become dilated and more distinct than natural; others are of new formation. All such newly formed channels originate from the pre-existing capillaries of the part. Their formation is described as follows:—One of the cells forming the boundary wall of a capillary throws out a protoplasmic process. This joins a similar process from another capillary, and the two vessels are thus united by a band of protoplasm, which, although at first solid, is subsequently hollowed out and permits the transit of blood.

The origin of the cells which are found in the inflamed tissues is a matter about which there has been much dispute, but may now be considered settled. They originate from two sources: (1) The leucocytes, whose diapedesis has already been described. These increase in number by the escape of fresh corpuscles from the vessels of the inflamed area, and also by fission and karyokinesis;

(2) The connective-tissue cells, which soon begin to appear at the seat of lesion. The fixed connective-tissue cells increase in size and multiply in number both by direct and indirect cell division. The wandering cells migrate to the area of inflammation and also multiply *in situ*. The leucocytes are present in such numbers as to obscure the other cells, and they constitute the so-called "round-cell infiltration"; but at the periphery of an inflamed area, and as the leucocytes disappear, the presence of the connective-tissue cells becomes evident, so that Cohnheim's view, that all the cells found in inflamed tissues are derived from the blood, must be given up. The commonest leucocyte found in the round-cell infiltrations is the ordinary so-called "multinuclear" or "neutrophile" white corpuscle.

In non-vascular tissues, such as the cornea, it has been shown that the cells found in its substance when it is inflamed are mostly due to an infiltration of leucocytes from the surrounding parts, and to a very slight extent only to the multiplication of the corneal corpuscles. Everywhere the round cell infiltration is chiefly made up of white corpuscles, and the connective-tissue cells undergo proliferative changes, and keep themselves in readiness for the later stages of inflammation and the regeneration of injured structures.

EXPLANATION OF THE CHANGES WHICH OCCUR IN INFLAMMATION.

The dilatation of the vessels and the increased afflux of blood which mark the earliest stage of the inflammatory process are due to the direct effect of the injury upon the vessels themselves. This dilatation is not a reflex change due to irritation of afferent nerve-fibres, for it can be caused by injury after the section of all nerves connected with the damaged part. It is due to an alteration in the tone of the muscular walls, and the injury acts either directly upon the muscle itself, or else upon the nerve filaments which it contains.

The retardation and ultimate stasis of the blood-

stream are the consequences of changes in the capillary blood pressure and in the vessel-wall whereby the relations between the blood and the vessel become altered. As a result, there is an increased resistance to the passage of blood through the vessel, a tendency to adhesion of the white blood-cells to the vessel-wall, and transit of greater numbers of them into the surrounding parts.

That these phenomena are due to changes in the vessel, and not to an alteration of the blood in the inflamed area is thus proved:—If the ear of a rabbit be deprived of blood for twenty-four hours, and the circulation then restored, it is found that, in consequence of the cutting-off of the blood-supply, the walls of the smaller vessels have become so much altered that all the phenomena of inflammation ensue, including dilatation of the arterioles and capillaries, followed by stasis and exudation. On the other hand, if, when stasis has commenced in an inflamed tissue, the engorged vessels are mechanically emptied of their contents, the white blood-cells which adhered to their walls traverse the rest of the circulation in a natural manner, and the rouleaux of red blood-cells break up, whilst, at the same time, if fresh blood be allowed to enter the inflamed area, stasis again occurs.

The **exudation** of fluid which accompanies inflammation is due to several factors. Serum passes out through the lymphatic and vascular capillaries into the substance of the surrounding tissue. The endothelial lining becomes affected in some way or another, and allows the exudation to filter through, but at the same time there must be some change in the capillary pressure, and also chemical changes in the tissues, to favour the osmotic current from the capillaries and lymphatics. It is assumed by some that the endothelium of the lymphatics is an actively secreting membrane, and that certain chemical or irritating substances act as local or general lymphagogues, so that an increased exudation would appear quite apart from other physical causes.

It is thus evident that the chief phenomena of inflammation are due to the **effect of the original injury upon the vessel-wall,** and the endothelium of the lymphatics and capillaries.

In all acute inflammations, however, there is another, and from a surgical point of view a most important, factor in the retardation of the circulation in an inflamed part. This is the **tension caused by the exudation.** The more fluid that escapes from the vessels, and the greater the rapidity with which it exudes, the greater will be the pressure on all the more yielding structures around. Further, the more resisting the tissues, and the less capable they are of swelling so as to make room for the exuded material, the greater will be the tension. As a consequence, the flow of blood in the vessels may be seriously retarded, and in some cases the retardation may be sufficiently extensive to cause the death of the surrounding parts. This is known as "sloughing" or "necrosis."

THE CLINICAL SIGNS OF INFLAMMATION

are swelling, heat, redness, and pain. Their occurrence may be briefly explained by reference to the pathological changes already described.

The **swelling** is the direct result of the increase of fluid in the part. The fulness of the vessels alone would account for part of the swelling, but most of the latter is due to the exudation of serum and liquor sanguinis.

The **heat** and **redness** are alike due to the increased vascularity of the inflamed area, and to the consequent transit of a larger quantity of blood than is natural. The increase of heat is always relative. The inflamed part is hotter than the corresponding portion of the body on the opposite side, but is never hotter than the blood itself, or than the mouth or the rectum. It is true that the local temperature may be raised to 100° or 101°, or higher, but when such is found to be the case, it will be found also that the general body-temperature is raised to a point at least as high.

Pain is due to pressure on, and stretching of, the peripheral nerves. It is always in direct proportion to the *tension* in the inflamed structures. Thus, in a part which can easily swell—*e.g.*, in the loose cellular tissue of the scrotum or of the eyelids—there is but little pain; but when inflammatory exudation occurs beneath tense structures, such as tendon sheaths, tight fasciæ, periosteum, &c., pain is always very intense.

TERMINATIONS OF INFLAMMATION.

The inflammatory process may at any stage undergo **resolution**. The extent to which it will progress depends, as will presently be shown, upon the nature of the injury, and the presence of decomposing material or of persistent irritation.

If resolution occur early, the hyperæmia may pass away even before stasis is reached; if later, the stasis may be broken up, the vessel may return to its natural condition, and the blood-stream may again resume its natural flow. If serum has been exuded into the tissue, it may again be absorbed by the lymphatics. If fibrin has been formed, it may be disintegrated and removed by leucocytes.

The leucocytes themselves frequently disappear, but in other cases they remain in the tissue, and in yet others, to be alluded to immediately, they may collect in sufficient quantities to form **pus.**

If the leucocytes remain in the tissue, they do not remain as such. They assist in the process of resolution, clearing away noxious and necrosed substances, or they degenerate. The majority of leucocytes possess the power of ingesting and digesting or destroying matter with which they come in conflicting contact, and, since in most forms of inflammation as met with in surgical practice, pathogenic micro-organisms are found, white blood corpuscles containing bacteria will be seen almost invariably. The organisms are thereby absorbed, and thus the cause of inflammation is removed. The process is called **phagocytosis.** The leu-

cocytes are assisted in their work as scavengers by other wandering cells, which are always found in the connective tissue, and also by the fixed connective-tissue cells themselves. It seems, however, that the leucocytes act also chemically, destroying or neutralising the irritant without being able to ingest it; as, for example, when the irritant is a chemical substance in solution (see "Suppuration," Chapter V.). Many or most of the leucocytes undoubtedly degenerate with resolution, and it is possible that they may also serve as food for the connective-tissue cells destined to regenerate the injured structures. It was thought until recently that the white corpuscles themselves built up the scar tissue; *i.e.*, that they became elongated or spindle-shaped, after a time fibrillated, and eventually fibrous or connective tissue. Most authorities agree now, however, that though very occasionally perhaps this may occur, yet in warm-blooded animals the scar tissue is formed by the connective-tissue cells. Resolution and scarring only occur where the cells receive a sufficient blood supply for their nutrition, &c.

Theories of Inflammation.—The chief phenomena of inflammation have been considered above, and a partial explanation of them has been given. The essence or nature of inflammatory processes is still obscure, and various theories have been held from time to time, of which some may be briefly considered without entering into any discussion regarding their value or adequacy. (1) Hyperæmia was regarded to be the leading phenomenon of inflammation, and hence the cause of inflammation was found either in a paralysis of the vasomotor nerves or in a constriction of the injured arteries followed by congestion of the surrounding areas. It became evident, however, that there may be hyperæmia without inflammation. (2) Virchow therefore explained inflammation as being caused by an increased nutritive and functional activity and proliferation of the cells in the affected tissues. He assumed that the connective-tissue cells multiply rapidly, and thus lead to the round-cell infiltration. Pus accordingly was considered

to be the outcome of proliferating connective-tissue cells, the leucocytes taking practically no part in its formation. (3) This conception was soon modified by Cohnheim, who showed that the round-cell infiltration consists almost entirely of white blood corpuscles which have passed out through the injured vessel-wall. The latter, acted upon by the irritant, becomes more permeable, and allows exudation and diapedesis to take place. During the process of repair the leucocytes were assumed to be gradually converted into fibrous tissue. He showed that in non-vascular tissues even, as the cornea, the round-cell infiltration is due to a migration of the white corpuscles from the vessels in the conjunctiva, or the vascular borders of the cornea. It has since then been confirmed by most observers that the round-cell infiltration and the pus cells are almost entirely made up of leucocytes, but that the latter, in warm-blooded animals at least, do not become converted into fibrous tissue, as Cohnheim assumed. The cicatricial repair is brought about by the connective-tissue cells, which undergo rapid division, and assume an increased activity. (4) Recently, Metchnikoff, on the strength of comparative observations on lower animals, has propounded a fascinating theory which seeks the first principle of inflammation in the phenomena of phagocytosis. These various views, briefly indicated here, cannot be considered at length in such a work as this. Inflammation is a complex process, the nature of which is still obscure, and depends on a series of causes and phenomena. Nervous influences no doubt play also an important part, for it has been shown that after section of the sympathetic nerve, irritation often produces in a part an extremely severe inflammation, which passes off slowly, though at other times such section seems to lead to a quick resolution of a pre-existing inflammation.

CATARRH.—CATARRHAL INFLAMMATION.

Catarrh is a form of inflammation affecting epithelial surfaces, and, although more common in mucous membranes,

occurs in the skin as "eczema." All catarrhal inflammations are characterised by the comparatively slight changes which take place in the epithelial cells, whilst the deeper tissues are involved in the destruction which is seen in all inflammatory processes. Catarrh may be excited by mechanical injuries, by exposure to cold or wet, or to irritating matters, as well as by certain constitutional contions such as gout and scrofula.

In the early stage of catarrhal inflammation there is redness and hyperæmia, and then a serous exudation into the sub-epithelial cellular tissue, quickly followed by oozing of the fluid amongst the epithelial cells, and its escape on to the surface.

In many cases no further change ensues in the connective tissue, but in some a plastic exudation, with formation of fibrin, results, and the affected part becomes more swollen and indurated, whilst in others the inflammation proceeds to suppuration, and pus-cells force their way between the epithelial elements, and are discharged from the skin or mucous membrane affected.

In the meantime, the epithelial cells themselves, being more tough and resistant than the softer sub-epithelial tissues, appear to derive actual benefit from the unusual vascularity of the deeper structures and the consequent increased supply of nourishment. They multiply with unusual rapidity, and, in the case of mucous membranes, secrete much more mucus than in their natural state; their secretion is necessarily mingled with the serum which exudes from the vessels below, and thus forms the copious watery and sticky discharge characteristic of mucous catarrh. In this discharge are found a certain number of leucocytes and epithelial cells in varying numbers and of different shapes and sizes. When suppuration occurs, and the discharge becomes muco-purulent or purulent, many of the pus-cells may be seen to have made their way into the substance of the epithelial cells, and appear to have been formed by multiplication of the cell-nuclei, an appearance

which is a deceptive one. If the inflammatory process continues, the epithelial cells are in time destroyed and an ulcerated surface is exposed. In such a case the inflammation ceases to be catarrhal.

Post-mortem examination shows but little alteration in cases of acute catarrh, for the hyperæmia and swelling quickly subside after death. In cases of chronic catarrh, however, there is much pigmentation, which results from the exudation of red blood-cells and disintegration of their colouring matter. This is well shown in cases of chronic cystitis, or during life in the legs of those who suffer from chronic eczema.

CHAPTER V.

SUPPURATION.

Suppuration, or the formation of pus, is the final result of all acute inflammations in which neither resolution nor repair occurs. The pus may be contained in a cavity forming an abscess, it may be diffused, or may be discharged from a free surface, part of which has already been destroyed, and which is said to be in a state of ulceration.

ÆTIOLOGY OF SUPPURATION.

It has already been said that any injury may cause inflammation, but the essential characteristic of a simple traumatic inflammation is that *it is strictly localised to the seat of injury, and does not progress after the cause is removed.* A transient injury, therefore, however severe, will not cause an inflammation which will progress to the formation of pus, for, before that stage has been reached, resolution will have taken place.

There are two chief causes for the progression of inflammation to suppuration. They are—first, **the presence of organisms**; second, **persistent irritation of the inflamed part**. These conclusions are based chiefly upon experiments, to some of which brief reference may be made.

In order to show that, even after the most severe injuries, suppuration does not necessarily ensue, Professors Hueter and Hallbauer destroyed portions of the muscle of a rabbit's thigh by either the actual cautery or by chloride of zinc. They found that, when antiseptic precautions

were employed, scarcely any vascular disturbance took place, and the dead tissue did not act as an irritant.

Other experiments by Professor Chauveau took matters a good deal further. This experimenter showed that the subcutaneous twisting of the spermatic cord in animals, which is practised in France instead of castration, always results in the complete death of the testis, and that under ordinary circumstances the dead organ does not act as an irritant towards the tissues in which it lies. The operation, which is known by the name of *bistournage*, is performed by merely seizing and twisting the testis four or five times, the result being occlusion of the spermatic artery.

But although, under ordinary circumstances, no suppuration ensued, Chauveau showed that, if any septic matter was in the testis at the time of operation, pus was formed, and the dead organ was cast off. This was demonstrated as follows:—Some of the fluid from a septic abscess containing organisms was injected into a vein before the operation was performed. The organisms being thus circulating through the testis at the time that the cord was twisted, some of them necessarily remained in the organ after the operation, and in all cases where this injection was practised it was found that the tissues became filled with pus which contained numerous organisms. It then remained to prove that the suppuration was due to the presence of the micro-organisms in the testis, and not to the general contamination of the blood—a problem which was solved by performing the operation of bistournage *before* injecting the septic material, by which means, the spermatic artery having been occluded before the organisms were introduced, none of them could subsequently enter it. In such experiments no suppuration occurred, and it was therefore concluded that the presence of micro-organisms at the seat of injury alone was sufficient cause for suppuration.

But not only does suppuration result from an injury when the septic material is directly injected into the blood, or enters at a wounded surface; it may also result from the

ingestion of putrid liquid by the stomach and intestinal tract. Professor Kocher has shown that, in dogs, wounds of bone which are in a perfectly healthy condition may be made to suppurate, and diffuse osteitis may be caused by feeding the animal on putrid material, and there can be no reasonable doubt that such a result implies that micro-organisms introduced through the intestinal tract may contaminate a wound, and that thus the general condition of a patient may exercise much influence on the healing process.

This is abundantly illustrated by clinical observations; for not only do wounds seldom heal well when the patient is suffering from such diseases as pyæmia, erysipelas, &c., but in many patients in bad health, injuries such as slight contusions are followed by suppuration even in cases where no skin wound exists.

Although, however, as is shown above, it is proved that organisms have much to do with the causation of suppuration, it must be clearly understood that most of the organisms which are concerned with the formation of pus are powerless in the presence of healthy tissues, and it has been shown that if injected into the blood they are quickly destroyed. It has already been mentioned that rest is essential for the growth and activity of most of the bacteria, and the blood is therefore no favourable soil for these parasites. But it is evident that in the case of an injury these conditions are materially altered, for as the direct result of injury there is escape of blood from the damaged vessels, and thus a stagnant fluid provided; and as a consequence of the succeeding inflammation, there is an exudation of more fluid, and an impairment of the vitality of the structures involved. Should bacteria therefore come into contact with injured parts, they are placed under much more favourable conditions for growth than obtain in the healthy body.

It is to the same tendency of organisms to live in injured, unhealthy, or dead tissues that the suppuration of lacerated

wounds is to be attributed, for in all of these there are to be found certain minute portions of tissue whose vitality has been lowered or destroyed, and it is in these that putrefactive processes commence.

Thus, it must be added that **the nature of the injury** is of considerable importance in deciding the question as to whether suppuration will or will not ensue upon its infliction.

The advent of organisms to a previously healthy wound is indeed always the sign for an immediate increase of the inflammatory process, an increase which is almost certainly the result of the action of the bacteria and their chemical products on the damaged and dead tissues and fluid in which they grow, and the consequent formation of irritating chemical products. The increase of the inflammation is, in fact, designed to rid the wound of the parasitic growth, and as the leucocytes are to a great extent the agents by which the bacteria are destroyed when under natural conditions they obtain access to the body, it is clear that the increased flow of blood, the exudation of cells, and the formation of interstitial granulation tissue are really reparative and not destructive. But in this struggle with the invaders many leucocytes die, and it is the living leucocytes which appear in increased numbers at the seat of lesion, together with the dead leucocytes and the fluid in which they lie, which constitute pus.

There is, however, another cause for the progression of any inflammation to suppuration—namely, **irritation**.

The inflammation set up by any transient injury will subside in the absence of septic infection, but, if the cause of the inflammation be allowed to remain, if the injury be, so to say, long-lasting, if in fact, persistent irritation be present, then the inflammation will not be allowed to subside; it will progress, more and more exudation will take place, and pus will finally be formed. This has been well demonstrated by Chauveau, who found that if, after practising bistournage, the testis and surrounding parts

were repeatedly manipulated, inflammatory changes resulted, and suppuration ensued, though the pus in such cases did not contain any micro-organisms. Similarly, in man, even in aseptic wounds, or in the case of damaged tissues which have never been exposed by any skin lesion, suppuration may ensue upon want of rest, or irritation by some foreign body, the pus in such cases not containing organisms and possessing hardly any irritating qualities. Thus, suppuration may occur in cases of simple fractures not kept at rest, or after the injection of irritating liquids, such as iodine or strong carbolic acid, notwithstanding the aseptic nature of such fluids.

It has already been mentioned that under natural conditions suppuration is a reparative process, the living cellular constituents of pus appearing to be actively engaged in destroying the organisms which either caused or keep up the suppuration. But besides these phagocytic elements in pus there are found also chemical substances of great power well suited to assist the tissues in their struggle for repair, for, from pus a digestive proteolytic ferment can be separated, and Leber has also shewn that whilst metal wires inserted into the anterior chamber of a rabbit's eye will readily cause suppuration, the pus thus formed will gradually dissolve the metal. We have then in pus—(*a*) phagocytic cells, (*b*) digestive ferments, and (*c*) a strong chemical agent of great destructive power; and this will explain readily the rapid dissolution of tissues caused by acute suppuration as well as the destruction of the bacilli.

From the foregoing experiments, therefore, it may be considered proved—

First, that the most severe injuries do not of themselves cause suppuration in healthy tissues.

Second, that if the part injured be exposed in any way to "septic" influences, suppuration will ensue.

Third, that if the part injured be exposed to irritation, suppuration will ensue.

PYOGENIC ORGANISMS AND THEIR MODE OF ACTION.

The commonest causes of suppuration are undoubtedly micro-organisms. Of these, a few are so commonly found in pus that they have been singled out as *pyogenic* schizomycetes. It must be remembered, however, that many other cocci and bacilli, under special conditions or in exceptional cases, may produce suppuration, and again the so-called pyogenic germs occasionally may fail to produce suppuration or may lead to other lesions. The pyogenic organisms therefore cannot be considered to be specific organisms in the sense defined above. Most of the schizomycetes found in pus belong to the group of micrococci. The commonest pyogenic cocci are the—(1) Staphylococcus pyogenes aureus, (2) Staphylococcus pyogenes albus, (3) Streptococcus pyogenes; besides these, the following are not infrequently found: the Staphylococcus citreus, the Staphylococcus aureus flavus and albus, the Micrococcus tenuis, and the Bacillus pyogenes fœtidus. The Pneumococcus has been discovered in many cases of purulent otitis media and meningitis; recently the Bacillus coli commune, a normal inhabitant of the intestines, has been obtained in various forms of abscesses.

The pyogenic cocci are apparently present in the dust around us, and it is possible that the Staphylococcus epidermidis albus, which is a normal inhabitant of the skin or cutaneous glands, is merely a variety of, or identical with, the Staphylococcus pyogenes albus. In pus also an interesting bacillus is not infrequently found—viz., the Bacillus pyocyaneus. The latter in artificial nutrient media produces a blue or bluish-green pigment, which it also develops in pus (blue pus).

If the pyogenic cocci *i.e.*, the Staphylococcus pyogenes aureus and albus and the Streptococcus pyogenes—gain access into the circulation, septicæmia or pyæmia is the result

(see Chapter VIII.). Again in ulcerative endocarditis they are commonly found, and the Streptococcus pyogenes is apparently identical with the streptococcus of erysipelas. It is important to keep these facts in mind, because they explain many accidents and calamities in clinical surgery. The most important question to answer is, "How do these cocci produce or lead to suppuration?" It is certainly not their mere mechanical presence, but the action of their chemical products, which is responsible for the production of pus. Thus, it has been shown that sterilised filtered cultures—*i.e.*, cultures freed from the microbes, but containing their products—will produce suppuration when the living organisms fail to do so; and Leber has succeeded in separating crystalline bodies from cultures of staphylococci which were highly pyogenic. It is evident, therefore, that the production of pus is finally always due to some irritant, and that in this respect, after all, bacterial and non-bacterial suppuration follow the same law.

All these pyogenic cocci are often harmless when in contact with healthy tissues, but when they gain access to a portion of the body whose vitality has been lowered or destroyed by injury, or when the individual itself is in a weak or debilitated condition, they are able to exercise their undoubted power of exciting suppuration. Thus, it has been shown that the Staphylococcus pyogenes aureus injected into the circulation of a rabbit in most cases will produce no ill effects, but if the cardiac valves have been previously injured, an infective endocarditis will result; in other instances the simultaneous injection of a sugar solution has enabled the Staphylococcus pyogenes albus to show its pyogenic power, and numerous other experiments have clearly demonstrated the inability of injured or unhealthy tissues to protect themselves.

FORMATION OF PUS.

In describing the changes that occur in inflammation, it has already been noted that the white blood-cells accumulate in the softened tissues, and that numerous new blood-vessels run amongst the newly formed cells. Both cells and vessels are bathed in the exuded serum, and matted together by the coagulated fibrin. This vascularised tissue, rich in cells, is often called **inflammatory new formation**, and sometimes **interstitial granulation tissue**. If the inflammation progresses, the cells at the focus, or centre, of the inflammation increase in number, the tissues in which they lie are gradually destroyed, and finally a space or cavity is formed, bounded by the surrounding "interstitial granulation tissue," and containing leucocytes floating in a serous fluid. This fluid, with its contained cells, constitutes pus. The cavity containing it is called an abscess cavity, and the walls of the abscess cavity are formed by the "granulation tissue" already mentioned. An abscess once formed increases in size by a continuance of the process by which it was originally developed. Around it is an area of inflammation extending for a variable distance. This inflammation spreads, and, as the inflammatory process progresses to suppuration, the abscess grows larger by a gradual liquefaction of the tissues around it. In all cases the pus tends towards the direction of the least resistance, and thus, a free surface being reached, the pent-up fluid finally escapes. An abscess which forms rapidly, and which is accompanied by all the signs of inflammation, is called an "**acute abscess**," and it has been shown by Professor Ogston that all such abscesses contain micrococci, which have, in cases where there is no wound, presumably reached the inflammatory area through the medium of the blood. The pus of an acute abscess is hardly ever re-absorbed. It is practically dead tissue in a state of decomposition, and, acting as a foreign body to the parts around, prevents the inflammation from subsiding.

DIFFUSE SUPPURATION.

is the term applied to an inflammatory process going on to the formation of pus which is not limited by the walls of any definite cavity. In such a case the diffusion may be the result of a very widespread injury, *e.g.*, general contusion of a whole limb; but in many instances it is due to septic organisms of more than usual virulence, and gradually passes into the specific inflammations known by the names of "cellulitis" and "phlegmonous erysipelas." In cases of diffuse suppuration there appears to be but little tendency to the coagulation of the fibrin, which is the chief agent in limiting any collection of pus, and there is an almost complete absence of the formation of granulation tissue.

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SLOUGHING AND GANGRENE.

In all inflammations the vitality of the tissues is lowered, and as suppuration progresses they die. In most cases the death is molecular, and the portions of dead tissue are so minute that, even if cast off in the discharges, they are not noticeable. More generally, however, the small dead particles are dissolved in the exudation—most probably by the leucocytes—and it is only in inflammations of great intensity, or where the inflammatory products are under great tension, that larger portions of the inflamed structures perish. Such dead portions are called "sloughs," and when still larger the part is said to be "gangrenous." The dead tissues are subsequently separated from the living by a process of ulceration.

CHRONIC ABSCESS.

A chronic abscess is one which is of slow growth, in which the signs of inflammation are but little marked. Such abscesses result either from long-continued irritation, or from an injury inflicted upon unhealthy structures. The process by which the pus is formed does not differ from that which occurs in acute suppuration, but, on account of

the long continuance of the inflammation many of the proliferating connective-tissue cells near the boundary of the inflamed area remain in the part long enough to undergo further development, and thus the abscess-wall is liable to become greatly thickened by newly formed fibrous tissue. In some cases this thickening is so considerable that the pus becomes enclosed in a definite capsule, to which the name of "pyogenic membrane" has been applied. The pus of a chronic abscess in a healthy person does not differ from that of an acute abscess, except in that it often contains no micrococci. The latter is a fact to be remembered, for so long as all septic influences are kept away from the abscess-wall, so long will the formation of pus remain slow, and so long will signs of acute inflammation be absent. If, however, such an abscess be opened without proper precautions, and "septic" matter be introduced, putrefactive processes will be set up, an acute inflammation of the whole abscess-wall will ensue, and pus will be formed in greatly increased quantities. Although, however, neither micrococci nor the organisms which cause acute suppuration are to be found in the pus of a chronic abscess, yet most of such abscesses do depend for their origin on the presence of a bacillus, for a very large proportion of them are tubercular, and associated with the growth of the tubercle bacillus. It is further probable that in other cases the abscess was primarily due to the action of pyogenic germs which died at a later stage, for it has been shown that pus is an unsuitable soil for at least some few organisms.

Chronic abscesses often attain great size, and may remain for many months or even years without undergoing any material alteration in size. In some cases, and especially when the exciting cause has been removed, the fluid portions of the pus may be absorbed, and the pus-cells may dry up and become converted into a caseous mass, part of which may subsequently become calcified. When absorption is in progress, the contents of a chronic abscess present varying degrees of inspissation.

Occasionally, when the contents of an abscess have been apparently absorbed, there is a recurrence of inflammation after an interval which may amount to years, and pus is again formed. To such abscesses, occurring in the site of past suppuration, the term "residual" has been applied by Sir James Paget.

LYMPHATIC ABSCESS.

Lymphatic or "cold" abscesses are sometimes separated from the chronic variety on account of the greater rapidity with which the formation of pus in them takes place. They may indeed form very rapidly, and a case is recorded by Brodie in which he removed three quarts of pus from the thigh of a gentleman in whom it had collected in the space of three weeks. Such an abscess could scarcely be called "chronic," yet it differs from an acute abscess in the complete, or almost complete, absence of pain, redness, and heat. The walls of such abscesses are usually very thin, and their pus is often ill-formed and watery. It is probable that many lymphatic abscesses are of tubercular origin.

HEALING OF AN ABSCESS.

As already mentioned, the pus of any acute abscess is practically dead and "septic" material, and, as such, acts as a foreign body to the tissues around it, and keeps up the suppuration. When, however, the contents of such an abscess have been discharged, and nothing remains to cause a continuance of the inflammatory process, the abscess-cavity is rapidly obliterated. This obliteration occurs as follows:—The granulating surfaces of the abscess-walls are brought into contact by the pressure exercised on them by the surrounding tissues, which have until now been mechanically distended by the collection of pus. This pressure is naturally greatest in the deepest parts of a wound, and thus it is in its deepest parts that the walls of an abscess are naturally first approximated. Such an approximation soon results in an actual growing together of the opposing

layers of granulations, and a gradual obliteration of the abscess-sac from below upwards. If, however, the walls of the abscess are very thick, or the surrounding tissues are much matted together, the collapse of the abscess-sac will not take place, and pus will continue to be formed from the granulating surfaces. But it does not follow that even in these circumstances healing will not occur, for the abscess-cavity may be in great part filled by a growth of fresh granulations and fibrous tissue, as in the process of healing of an ulcer. The slowness with which this process occurs, however, is well illustrated by the great length of time required to fill up the cavity of an abscess situated in an absolutely unyielding structure, such as bone, for here, the collapse of the sac being impossible, its walls cannot directly grow together.

In some cases an abscess does not altogether close, but, contracting to a narrow suppurating track, continues to discharge pus. Such a track is called a **sinus**. Sinuses are often long, narrow, and tortuous. They are lined by ill-formed granulations, and secrete thin, watery pus. Their walls are commonly thickened by fibrous tissue, and a bunch of granulations commonly sprouts from the orifice. Anything which keeps up irritation will cause a sinus. Amongst the commonest causes are the presence of foreign bodies, such as dead bone, portions of clothing, &c.; the retention of pus in an ill-drained cavity; constant movements by neighbouring muscles; and the passage of irritating matters, such as fæces or urine.

ULCERATION.

Ulceration is the term employed to indicate an inflammation of a free surface which has progressed to suppuration. The process by which an ulcer is formed does not differ in any way from that by which an abscess is developed. In each case, an inflammation having been started, exudation, formation of new blood-vessels, and softening of the surrounding tissues occur, and in each granulation tissue is

developed and pus is formed. In the case of the abscess this pus cannot at once escape, for it is surrounded by granulation tissue on all sides; but in the case of an ulcer, the inflammation having occurred on a free surface, the pus escapes as quickly as it is formed, and the granulation tissue is found only in the "base" of the ulcer. It cannot be too clearly enforced that the section of the base of an ulcer and that of an abscess-wall possess precisely the same structure. Each is composed of "granulation tissue," and by each pus-cells are formed. If an abscess-cavity could be laid out on a free surface, it would form an ulcer; or if one ulcer could be placed over another so that the pus from each was shut in, a cavity lined by granulation tissue and structurally identical with an abscess would be formed. Ulceration is commonly said to mean "molecular death," and this is true. But it is also true that suppuration means precisely the same, and that the gradual destruction of tissue already described in dealing with the formation of an abscess differs in no way from the "molecular death" of ulceration.

An ulcer, like an abscess, may result from persistent irritation, or from a septic condition of a wounded surface, In the case of a traumatic ulcer of the skin, the initial injury is often slight, perhaps an abrasion destroying the surface epithelium and exposing the papillary layer. Such a wounded surface would readily heal if not exposed to irritation by dirt or mechanical stimuli; but, if so exposed, the inflammation started by the injury progresses through the stages of hyperæmia, stasis, exudation, &c., and results in the formation of granulation tissue from which pus-cells are thrown off. If the irritation be kept up, or if septic conditions exist, the ulcer may spread, and the deeper structures will be gradually involved and will perish in the process of suppuration. After a time the ulcer ceases to extend, and either remains stationary or progresses towards cicatrisation. Its stationary condition is unnatural, and is usually the result of a continuation of the irritation, but

it may be due to various local conditions to which allusion will presently be made.

In describing any ulcer, attention must be paid to—

(1) The shape and extent;
(2) The floor and secretion;
(3) The edges;
(4) The surrounding parts.

Following this arrangement, we may now proceed to describe a **healthy, healing** ulcer—*i.e.*, one from which all irritating causes have been removed, and which is in process of cicatrisation.

(1) The **shape** and **extent** vary in different cases.

(2) The **floor** is on the same level throughout, and is formed of innumerable "granulations," which give it a velvety appearance.

Examined more closely, each granulation is found to consist of a small papilla composed entirely of white blood-cells loosely held together by fibrin, and having in its centre a single looped blood-vessel. All the granulations are of the same size, and are bright-red in colour. They are closely packed, and the whole base of the ulcer is evenly covered by them. Immediately below these granulations lie the tissues softened and irritated by the inflammatory process, containing exudation-cells and fluid, with many new blood-vessels and connective-tissue cells in a condition of active proliferation. The more deeply we pass the less marked are the signs of inflammation, and, after passing through the area of stasis and hyperæmia, we reach the healthy subjacent structures. In the deepest parts of the inflammatory area we find some newly formed fibrous tissue.

The **secretion** of a healthy ulcer is healthy **pus.** This is a thick, opaque, yellowish fluid of a creamy consistence, with a specific gravity of about 1030, and an alkaline reaction. If allowed to stand, it does not coagulate, but separates into two layers, the upper being clear and fluid, the lower dense and yellow. The clear fluid is almost

identical in chemical composition with serum. The denser layer is composed of "pus corpuscles." Examined microscopically, these are found to be round cells, generally finely granular, with a diameter of about $\frac{1}{2500}$ of an inch, with a lobed or multi-partite nucleus. They are escaped leucocytes, many of them showing active amœboid movement, and containing bacteria or cellular remains in their substance (phagocytosis). A large number of these white corpuscles are, however, dead, and being dead, have no amœboid movement.

(3) The **edges** of a healthy ulcer are not raised, or indurated, or sharply cut; they slope gently on to the base, and where the edges and base join there is a thin bluish-white pellicle marking the line of the advancing epithelium, which is eventually to overgrow the whole ulcerated surface.

(4) The **parts immediately around** a healthy ulcer are red and inflamed, but this inflammation is very slight, and limited to the immediate vicinity of the ulcer. The skin in the neighbourhood is healthy, and is not congested, eczematous, œdematous, or otherwise abnormal.

PERFORATING ULCERS.

These are ulcers whose formation is essentially the result of some disease of the nerves, and which are characterised by their tendency to extend in depth rather than in area. They are almost always found on the sole of the foot, and especially beneath the ball of the great toe. They commence with a corn-like thickening of the epidermis, which may precede ulceration for several months. The ulcer has a great tendency to extend into the subjacent metatarso-phalangeal joint and to cause its suppuration; but, if treated by rest, cleanliness, and the removal of pressure, the ulceration generally ceases to extend. Perforating ulcers commonly complicate tabes dorsalis, and may occur in the course of other diseases of the spinal cord, or in cases of peripheral neuritis and notably in leprosy. They are sometimes associated with sweating and anæsthesia of the

neighbouring skin. If the part be treated by amputation, the ulcer is prone to recur on the stump.

HEALING OF AN ULCER.

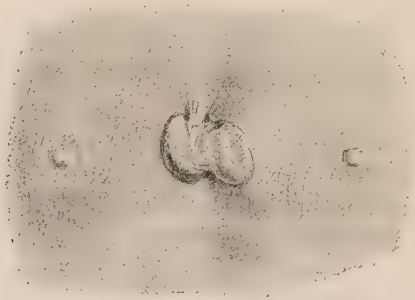
It has already been said that so long as an ulcer is irritated so long it tends to extend, but when all irritation has been removed, the inflammatory process becomes more limited, and **repair** commences. An ulcer implies loss of substance, and the first efforts of repair are directed towards filling up the gap made by the ulcerative process. Until this gap has been filled up—*i.e.*, until the base of the ulcer has been raised to a level with its edges and with the surrounding parts—the process of "skinning over" does not commence.

The gap is filled by fibrous tissue, which is formed from the surrounding proliferating connective-tissue cells. In the deeper parts of the granulations the newly formed and actively multiplying connective-tissue cells which lie amongst the exuded leucocytes show further activity, and, together with their neighbours, develop into fibres in the manner already described. Fresh granulations are now formed, the proliferation of cells continues, new loops of blood-vessels shoot up towards the free surface, and thus by a constant development of the cells in the deeper parts the ulcer, and an equally constant growth of fresh granulations, the base of the ulcer is raised to the level of the surrounding parts. When this is accomplished, the epithelial cells at the edges grow over the ulcerated surface, and gradually cover it in. At first the epithelial covering is very thin, and the granulating tissue beneath, shining through, gives it a bluish-white appearance. Later on, when the epithelium is thicker, and the subjacent structures have lost their vascularity, the "scar" or "cicatrix" is of a pearly-white colour.

It is the special characteristic of all newly formed fibrous tissue that it tends to contract, and, as a consequence, the scar tends to diminish in size for long after

the skinning-over process is complete. It is probable that this contraction is merely the result of lessened vascularity, for by the time that the epithelium has grown over the ulcerated surface, the numerous newly formed blood-vessels, having fulfilled their purpose of bringing up material to fill the gap, and of supplying the young connective tissue with nourishment, shrink and disappear. The young fibrous tissue consequently becomes more dry, and shrinks, and thus produces the contraction characteristic of all scar tissue. The result of this contraction is that the neighbouring parts tend to be drawn in towards the scar, and on

FIG. 5.



Keloid Scar on the Male Breast.

the extent to which they can be drawn in, even more than on the size of the original ulcer, the extent of the resulting cicatrix will depend. Thus, in extremely lax tissues, such as the scrotum and eyelid, large portions of skin can be removed with scarcely any visible scar resulting : but where the skin is tense—as, *e.g.*, over the front of the tibia—the scar of an ulcer is always relatively large.

When the whole thickness of the skin has been destroyed it is not reproduced, and the epithelial covering of the cicatrix is devoid of papillæ, hairs, sebaceous and sweat glands.

Occasionally the growth of scar tissue does not cease when

the wound has closed, but more fibrous tissue is produced, and a **keloid** growth results. Keloid is, indeed, a tumour composed of scar tissue, and may originate in the site of any healed wound, however minute. It is most frequently seen on the chest and shoulders, and is relatively common in the scars on the necks of scrofulous persons. The microscope shows that a keloid is composed of spindle cells and fibrous tissue, closely resembling the structure of a fibro-sarcoma. In most cases, after continuing to increase for an indefinite time, the growth of a keloid scar ceases, and in many instances the tumour already formed partially or entirely atrophies. Removal of keloid by operation is generally useless, as the scar of the operation wound also usually becomes keloid.

CHAPTER VI.

HEALING OF WOUNDS.

Although wounds are generally described as healing in four or five different ways, yet in all the process by which they are finally closed is the result of **inflammation**. It will be remembered that we have already defined inflammation (p. 29) as "the succession of changes which occurs in a living tissue *when it is injured*, &c.," and thus it may be said that every injury carries with it its own cure, for in all alike the healing process is of inflammatory origin, and is the direct result of the injury.

It is true that even within very recent years "healing by immediate union" has been described, but this is a process which may be said to have no pathology, for in it the several tissues are supposed to simply join without the intervention of any uniting material whatever. Thus, one piece of muscle would become attached to another piece, one end of a cut vessel to the other, and so forth. It is probable that this never really occurs, and that in all cases there is some intervening plastic material, however small in amount.

HEALING BY FIRST INTENTION

is healing without the formation of pus, and is best seen in cases of surgical operations, where healthy soft tissues have been cleanly divided by a sharp knife—*e.g.*, in operations for hare-lip or in flap amputations.

If in such a case all hæmorrhage is arrested, and the parts are brought into complete apposition, the following changes may be noticed :—

The edges of the wound at first become slightly red and swollen, the latter condition being often well demonstrated by the "burying" of the sutures which have been originally quite loosely tied, but which subsequently become tightened by the swelling of the soft parts. Following this there is some increase of heat and pain, and thus it will be seen that all the signs of inflammation are present, though all in a very slight degree, and strictly limited to the immediate neighbourhood of the wound. In the first few hours there is often a little oozing of serous or plastic fluid, but after the second day, or earlier, the pain, redness, heat, and swelling subside, and a thin red line is the only indication of the previous injury. Next, the redness of this line fades, and finally a narrow white streak or "cicatrix" remains.

A closer investigation of the process shows that in the absence of all sutures, the cut surfaces are within a few hours held together by some glutinous substance, and that with care they may be separated without the necessary causation of bleeding, the non-vascular uniting medium being fibrin, which has been exuded from the vessels in the immediate neighbourhood of the wound. Twenty-four hours later the uniting material has become vascularised, and any attempt to separate the cut surfaces will excite hæmorrhage, whilst, later still, the newly formed blood-vessels shrink and disappear, and the scar becomes absolutely non-vascular.

A microscopical examination of the wound affords an ample explanation of these clinical observations.

The immediate effect of the injury is to set up those changes which are characteristic of the inflammatory process, and consequently the parts in the immediate neighbourhood of the wound become the seat, first of hyperæmia, and then of stasis, exudation of liquor sanguinis and of leucocytes, proliferation of connective-tissue cells, formation of new blood-vessels, and softening of the inflamed tissues. In each flap there is a development of the

"inflammatory new formation" or "interstitial granulation tissue," already described, and between the flaps, and into the uniting fibrin, cells are exuded. These changes are quickly followed by the formation of loops of new blood-vessels and of granulation tissue between the flaps, which are in fact, as it were, melted into one another, in the same way as two pieces of sealing-wax may be united if heated and held in apposition, the inflammatory process softening the tissues in the same way as the flame softens the sealing-wax (Billroth).

FIG. 6.

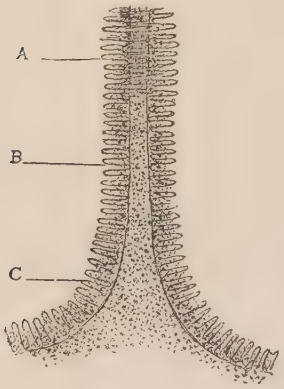


Diagram of a Healing Wound. At B the space between the flaps is filled with cells, which at A have become developed into fibrous tissue, and at C are accumulatiag in such numbers as to separate the flaps and form pus.

Now, it has already been shown that in healthy tissues no inflammation will progress to suppuration if the injured part is not irritated either by mechanical means or by the presence of septic material; so that in the wound in question, whilst the initial injury is alone capable of exciting a sufficient amount of inflammation to promote its healing, if no irritant be subsequently applied resolution will shortly ensue, and the inflammatory process will subside. If such be the case, the exudation of cells and liquor sanguinis, and the formation of new vessels will cease, for sufficient material has now been provided for the repair of the injured part. But the process of repair, begun chiefly by the connective-tissue cells, goes on until the scar is formed. Finally, both those cells which lie between the flaps, as well as those which lie in the softened tissues of the flaps themselves, will develop and form fibres, and finally the wounded surfaces will be united by a firm scar of non-vascular fibrous tissue.

Healing by scabbing.—Healing by scabbing is healing by first intention. The scab is formed by blood-clot, which is often held more firmly than it otherwise would be by being matted with hair and sometimes with other foreign bodies. The scab plays the part of a natural "dressing," and, by preventing mechanical irritation and infection by septic material from without, promotes healing without the formation of pus. In some cases the scab fails to prevent suppuration, and when it becomes separated a drop or two of pus is found beneath it, covering over an ulcerated surface—healing by first intention has failed, and union by granulation is in progress.

HEALING BY SECOND INTENTION, OR HEALING BY GRANULATION.

Healing by second intention is healing with formation of pus. If a wound about to heal by granulation is watched, it will be seen that the same changes occur in it at first as in the wound healing by first intention, but that, as time goes on, the signs of inflammation, instead of decreasing, become more marked. The redness and swelling extend, and the discharge, which originally consists of serum or of lymph, instead of soon drying up, increases in quantity and changes its character, for, though it is at first merely stained with the blood-colouring matter derived from disintegrating clots, it afterwards becomes thicker and whiter, and finally consists of pus.

At first, the edges of the wound are united by fibrin, but, as suppuration progresses, the pus gradually collects between the flaps, and after a time completely separates them, and exposes granulating surfaces.

Microscopically examined, the same changes are at first noticed as in healing by first intention. Instead, however, of the inflammatory process stopping short at the production of just a sufficient number of cells to unite the wounded surfaces, it progresses. More and more cells and fluid are exuded, the uniting fibrin, the interstitial granulation

tissue, and the softened tissues of the flaps become completely liquefied, and finally the flaps are separated, and their "granulating" surfaces are exposed. It will thus be seen that up to a certain point the processes of healing by first and by second intention are identical. The cause of the suppuration in the second case is to be found in the presence of either a mechanical irritant or of septic matter. Some foreign body may be in the wound, serum or blood may be retained under tension, or a portion of dead tissue may remain and decompose, and so the inflammation will progress instead of undergoing resolution.

A granulating surface being thus exposed, it in many cases proceeds to heal in exactly the same way as an ulcer—*i.e.*, by the development of fibrous tissue in the deeper parts from the connective-tissue cells, and by a constant formation of fresh granulations until the surface is reached. It is in these cases that phagocytosis can be well observed. The leucocytes are seen to contain numerous cocci, the organisms of suppuration. The connective-tissue cells similarly assist in this process, and, moreover, clear away the leucocytic débris, which remains behind after the death of the white corpuscles. Phagocytosis is decidedly a phenomenon of repair, which appears when the chief processes of inflammation have already passed away, or at any rate are declining.

In other cases healing by third intention takes place.

HEALING BY THIRD INTENTION, OR UNION OF GRANULATIONS.

This method of healing is really far more common than is usually supposed, and consists in the approximation and union of the opposed granulating surfaces, all irritating matter which originally caused suppuration having been first removed. Such a mode of union has already been described as taking place between the opposing walls of an abscess after evacuation of its contents, and constantly occurs in the deeper parts of all granulating flap-wounds,

though, on account of the fact that it cannot be seen in actual progress, it has scarcely obtained sufficient prominence.

Healing of lacerated and contused wounds.—In lacerated and contused wounds, not only is the initial injury greater than in clean cuts, but small portions of the damaged tissues are usually injured beyond repair, and die. These dead portions, being in contact with the living parts, and often infiltrated with septic material, act as foreign bodies to the tissues amongst which they lie and cause suppuration. On account of these reasons lacerated wounds cannot be expected to unite by first intention, for before healing can occur the dead parts or sloughs must be separated. After this has taken place, the wound heals by second or third intention in the manner already described.

THE EFFECT OF DRAINAGE OF WOUNDS.

In many large wounds it is customary to provide for the free escape of any fluids that may collect in the injured parts. This provision for the escape of fluid is spoken of as "drainage."

In what has been written above of the changes observed during the union of wounds, the escape of serous and plastic fluid, and in other cases of pus, has been mentioned as of usual occurrence. This fluid is derived in part from the contraction of clots and the squeezing out of serum, but in much greater part from the exudation which is the necessary accompaniment of all inflammation. In some cases, where large raw surfaces are left, as after amputations, the amount of this fluid is very considerable, and if no exit were allowed it might collect between the flaps, and, by mechanically separating them, and causing tension, would effectually prevent union by the first intention—a trouble which is obviated by the use of drainage-tubes during the first day or two after operation. But not only does retained fluid prevent healing by mechanically separating the parts; it also provides an admirable medium for

the development of any micro-organisms that may gain access to it.

It has been shown by Wegner that fluid containing micro-organisms may be introduced with impunity into the peritoneal cavity of an animal so long as no more fluid is used than can be readily absorbed. If, however, more fluid is injected than can be absorbed, it causes an accumulation in the peritoneal cavity, and peritonitis supervenes. The explanation of this is obvious. The unabsorbed fluid quickly becomes charged with animal matter by osmosis, and in this the micro-organisms rapidly develop, and cause putrefactive and inflammatory changes. When only a little fluid is used it is rapidly absorbed, and the micro-organisms coming into direct contact with living and healthy tissues, are immediately destroyed.

There is therefore a great difference between exposing to contamination an open granulating surface and one which is closed. Thousands of people, for instance, go about their daily work with large ulcers on their legs, freely exposed to dirt of all kinds, and septic in the extreme, yet no harm comes, for any organisms which may gain access to the ulcerated surface are either destroyed by the granulations—perhaps by phagocytosis or by the solvent action of the pus—or quickly washed away in the discharge, and get no opportunity of exciting putrefactive or other morbid changes. On the other hand, if but a few bacteria gain access to the retained fluid in an amputation wound, they will rapidly multiply in it, will cause decomposition of the fluid, and will form material which, if absorbed, will lead to severe constitutional symptons.

CHAPTER VII.

TRAUMATIC, SUPPURATIVE, AND HECTIC FEVER.

Traumatic fever is a fever due to an injury. Suppurative fever is a fever occurring in connection with the formation of pus.

TRAUMATIC FEVER.

If any severe injury is inflicted on a healthy person, the temperature, after a transient depression dependent on "shock," often rises above the normal, and continues to ascend for about twenty-four to forty-eight hours, usually attaining its highest point on the evening of the second day. The increased rapidity of pulse and respiration, the dryness of the skin and of the mouth, the furred tongue, constipated bowels, and scanty, acid urine, which are the usual accompaniments of fever, are all present. After the second day the temperature descends, and, as a rule, the fever has run its course by the fourth or fifth day. It never continues more than a week. Traumatic fever such as this has no connection with any septic condition of a wound. It is the direct result of changes set up by the injury itself, and, provided that nothing intervenes to alter its course, will reach its height and then subside in a perfectly definite manner.

The **causes** of this form of fever have been variously explained. The first and oldest theory is that it is due to the increased temperature of the parts which have become inflamed as the result of the injury. This, however, is not true, for, as has already been mentioned, the

increased temperature of the inflamed part is only relative, and never exceeds that of the blood. It is therefore not possible that the latter should become heated by its transit through the inflamed area.

The second theory is that the fever is caused by the absorption of the products of inflammation. Any substance which, when inoculated, causes a rise of temperature is said to be "pyrogenous," and it has been shown that the ferment which is present during the formation of fibrin belongs to this class. It is therefore probable that the absorption of inflammatory products independently of any septic contamination may cause fever.

The third theory is that the fever is of nervous origin—that the irritation of the peripheral nerves at the seat of injury, either mechanically or by implication in the inflammatory process, may reflexly cause the production of a greater amount of heat throughout the body. No doubt need be entertained of the possibility of such a mode of production of heat, for it finds a parallel in the normal physiological processes by which the temperature of the body is maintained at a uniform level, and it is certain that, in some cases at least, traumatic fever is of neurotic origin. Thus, in cases of simple fracture the temperature frequently rises within an hour or two of the receipt of the injury, when there has scarcely been time for the absorption of inflammatory products. And, again, in many other cases a splint, tightly applied, or inflicting pain, is the cause of an attack of fever, which immediately subsides after the removal or loosening of bandages or strapping. In another variety of traumatic fever, to which the name of "urinary" or "urethral" fever has been applied, the fever is the direct result of irritation of, or injury to, the urinary tracts, and especially the male urethra. In some patients the passage of a catheter, which in no way injures the mucous membrane, is immediately followed by a rigor and faintness, with much shock, succeeded by a rise of temperature of several degrees, which in its turn usually quickly passes away,

the attack being evidently throughout of a neurotic nature.

It is thus clear that traumatic fever runs its most typical course when a wound is healing by first intention, that it is independent of any septic changes, and may be, and often is, present in injuries unaccompanied by any open wound at all.

If a wound does not heal by first intention, and pus is formed, the traumatic fever, instead of subsiding, is liable to continue, and to pass gradually into suppurative fever.

SUPPURATIVE FEVER.

This form of fever, unlike that which precedes it, attains no definite height and runs no definite course, for, unlike the former, it is not dependent upon a single and transient cause, but is liable to last so long as the suppuration persists.

Cause.—The same causes that produce traumatic fever may also influence the course of suppurative fever, but the most important and most active cause of the latter is undoubtedly the **absorption of "pyrogenous"** or heat-producing, **material** from the wound. It has been already mentioned more than once that the cause of suppuration is either mechanical irritation or the presence of micro-organisms which are pyogenic or septic in nature. Now, the pus which is formed as the result of mere mechanical irritation has little more pyrogenous properties than has fibrin ferment, but the pus which is produced by the action of pyogenic cocci or is contaminated by septic material is in the highest degree pyrogenous. In speaking of the drainage of wounds, it has already been said that the retained products of inflammation form a most favourable medium for the development of micro-organisms, and if pus which is at first perfectly healthy is submitted to the action of the organisms capable of promoting suppuration or putrefaction, it will soon be found that the latter have produced in the previously almost innocuous pus morbid products which,

if injected, will cause all the symptoms of severe suppurative fever. Thus, it has been shown experimentally that the chemical products of micro-organisms are capable of causing fever when injected into the circulation or subcutaneously. The pyogenic cocci manufacture albumoses or albuminoid bodies which are markedly pyrogenous. Further it has been shown that the dead bodies of bacteria contain substances which are not only pyogenic but lead often to great febrile disturbances, accompanied by marked changes in the blood, both chemical and morphological. There is often a pronounced leucocytosis, and an impairment of the coagulative power of the blood. In some cases, no doubt, the fever produced is a defensive mechanism applied by the body to counteract the effects of intoxication or infection. In other cases, however, it signifies a gradual decline in the resistance of the animal organism.

If, however, a free exit is given to all the products of inflammation, first, they will not be absorbed at all, and, secondly, they will not remain sufficiently long exposed to the action of micro-organisms to become contaminated before being discharged from contact with the tissues capable of absorbing them.

It is thus evident that suppuration may occur, and large quantities of pus may be formed, and yet no "suppurative fever" may be present. The occurrence of the latter is due to the **retention** and **absorption** of altered inflammatory products rather than to their formation.

Just as traumatic fever fades into suppurative fever, so the latter fades into hectic fever.

HECTIC FEVER.

The special characteristic of hectic is the marked periodicity of the rise of temperature, which always attains its greatest height towards evening, and then, after perspiration more or less profuse, gradually sinks, sometimes becoming normal, but often remaining persistently a little higher than natural.

This form of fever is almost invariably the result of long-continued suppuration, but whether it owns the same or similar causes as traumatic and suppurative fevers, or whether it has an altogether different origin, cannot certainly be stated. Its pathology is as yet obscure, but it has been shown by experiment that many albumoses which are amongst the metabolic products of suppurative organisms, are capable of producing fever lasting for several days, and Koch's tuberculine, which to a great extent consists of toxic albumoses, when injected into the animal body, will cause a prolonged rise of temperature, so that we must believe that the hectic fever is due to a repeated absorption of peculiar chemical substances elaborated by the micro-organisms. These in some cases react as albumoses, but their true nature is at present unknown.

CHAPTER VIII.

SEPTICÆMIA AND PYÆMIA.

Septicæmia is a clinical term applied to a constitutional condition resulting from absorption of the chemical products of pyogenic or septic organisms, which runs its course without the development of secondary metastatic suppurations.

It has already been mentioned that the chief cause of suppurative fever is the absorption of the chemical products of suppuration or decomposition from a wound, which under such conditions is called septic, not because there are necessarily putrefactive or truly septic processes going on, but because those changes exist in it which render it unhealthy in appearance and dangerous to the patient. Septicæmia is to be regarded as simply a severe form of suppurative fever, which is also the result of absorption of toxic matter from an inflammatory area. The agents at work in causing decomposition in all animal substances are micro-organisms, and in septicæmia also the poisonous material results from the action of these organised ferments. It has, however, been shown by experiments on animals that two separate conditions exist which have previously been included under the one name of septicæmia, and before proceeding further it is necessary to elucidate this point.

In all decomposing animal fluids certain bodies are found, to which the general name of "ptomaines" has been applied, and one of these, to which the name of sepsin has been given, was supposed by its discoverer to be the sole cause of septicæmia. This supposition is no longer enter-

tained, for there is every reason to believe that the other basic bodies are quite as injurious, and, moreover, we know now that the ptomaines are less important than was at one time assumed, and that there are other chemical substances—toxines—apparently of greater activity, and present in larger quantity in decomposing fluids. If fluid taken from a decomposing wound, and therefore containing various kinds of toxines, be first of all carefully filtered and thereby purified from the numerous micro-organisms which it contains, and then injected into an animal, it is evident that the latter is inoculated, not with the organisms themselves, but with the chemical products of their action on animal matter. To the poisoned condition which results the name of **sapræmia**, or **septic intoxication**, is applied.

The chief symptoms of this are slight muscular twitchings and loss of power, with great restlessness, vomiting, and diarrhœa. The temperature rises, the breathing becomes difficult, and finally death ensues from cardiac weakness. Such is the course of events if a sufficiently large dose has been given, and, when an excessive quantity has been injected, the animal may die in an hour or two. When the dose is but small, recovery follows after a slight febrile attack. The course of events is entirely analogous to what is seen when any other active unorganised poison, such as strychnine, aconitine, &c., is administered. If the dose is large enough, the patient dies; if not, the poison is excreted, and he recovers.

In the case of Sapræmia, then, the process is pre-eminently intoxicative. A familiar example will show this clearly. Supposing we are dealing with an uterus immediately after delivery, and through neglect the clots and decidual remains are not kept in a clean and healthy condition, the result is a more or less raw surface, covered with dead and devitalised débris. This forms a suitable soil for **saprophytes, organisms unable to grow on or in healthy living tissues.** They will gain a footing here, and manufacture their toxines (ptomaines, albumoses, &c.). These substances are absorbed

into the circulation, and produce the typical symptoms associated with sapræmia. The saprophytic organisms, however, cannot spread into the healthier tissues of the uterus or the parts around; they cannot enter the lymph-channels or the circulation, because they are restricted to dead or dying matter for their growth. If the clots, &c., be removed by curetting or antiseptic treatment, the organisms are destroyed as well, and their soil is rendered unsuitable; and now, granted the dose of poison was not excessive, the symptoms will disappear. If, however, the dead tissues be not removed, the process of decomposition and necrosis will continue, and the saprophytes will find more soil, and, increasing in numbers, will elaborate more toxines, and the symptoms of intoxication will become severer. The latter are always proportional to the dose of poison manufactured and to the number of organisms present. Sapræmia, as such, then, is due to microbes—*i.e.*, it is an "infective process, a **saprophytic infection**," and as such it is also capable of transmission from one individual to another, for, given similar conditions, the organisms may be carried through carelessness and want of cleanliness to a suitable surface, and there set up identical changes. Nevertheless, we are dealing essentially with an intoxication. The poison which is absorbed is as incapable as is strychnine of multiplying in the body, and consequently, if a drop or two of the blood of the animal experimented on be injected into another animal, no effects are produced, for the quantity of the poison used is but infinitesimal.

Septic infection, or **true septicæmia,** differs from sapræmia in the essential particular that it is a true infective process **due to micro-organisms capable of living and multiplying in living tissues**—*i.e.*, that it is due to parasitic schizomycetes. These are commonly the familiar pyogenic micrococci. It is experimentally produced by injecting infected fluid containing such organisms into a susceptible animal. Saprophytes and the common organisms which are most active in producing decomposition are not the

cause of true septicæmia, for they are incapable of acting on living tissues, and in the presence of the latter are soon destroyed. It is certain, therefore, that other more active organisms, capable of living and multiplying in the tissues and blood—pathogenic parasitic organisms—are the cause of septicæmia. Koch has indeed shown that in the case of mice such an agent exists—namely, a very slender bacillus, which, if introduced into a mouse in the smallest quantities, multiplies with extreme rapidity, and quickly causes the death of the animal. If the point of a knife be dipped into the blood of a mouse which has died from this cause, and a slight scratch be inflicted on another mouse, the latter also dies with similar symptoms. Many other bacterial diseases met with in animals are truly septicæmic, as, *e.g.*, anthrax, pneumococcus-septicæmia, &c. Strictly speaking, then, in septicæmia there must be an infective agent, the pathogenic organism, which, introduced in however minute a dose into a susceptible body, soon multiplies in the tissue and blood, thus causing a general infection. The term septicæmia, however, as used in clinical surgery, includes other conditions besides these, and an illustration will make this clear.

Let us suppose that pyogenic micrococci find entrance into the uterine cavity after delivery, and begin to grow and multiply in the wall of the uterus. They would here produce their toxines, as the saprophytes did in the previous case, and, these poisonous substances being absorbed, a similar intoxication must result. The organisms, however, being parasitic in nature, will spread in and along the healthy tissues also, where the saprophytes could not live, and will pass through and beyond the uterine walls, so that soon there is an extra-uterine form of infection, and with it an additional production of toxines and an exacerbation of the symptoms of intoxication. The process in such a case probably spreads along the lymph-channels, and though there are all the symptoms of septicæmia present, the blood may be entirely free from micro-organisms, the symptoms being due to an intoxication,

just as in the case of sapræmia. In both there is an infection, in both there is an intoxication; but in the one case we have to deal with a "parasitic" infection, in the other with a "saprophytic" one.

But there are other cases in which the cocci, besides spreading along the lymphatics, may enter the circulation through the veins and be carried away to distant parts. They will then grow and multiply in the blood, and here also will manufacture their virus, which, being readily absorbed, will rapidly cause severe symptoms of intoxication. At the same time, they may be diffused also generally throughout the tissues by the blood-stream, so that practically the whole body becomes infected. An infection, therefore, may spread (*a*) along the lymphatics; (*b*) by the blood-stream, or (*c*) in both ways, and may be accompanied or followed by identical, or at any rate similar, symptoms, which clinically are included in the term septicæmia, though **strictly speaking only when the blood is infected should we speak of "septicæmia."** The poison, then, in septicæmia is essentially infective, and the symptoms do not solely depend, as in the case of sapræmia, on the amount absorbed from the wound, for, however small the quantity of the poison, the organisms which it contains are capable of indefinite multiplication in living tissues and in the blood itself, and will thus in time produce a sufficient amount of poison to cause death. The chief symptoms are extreme feebleness and languor, with gradual slowing of respiration. The animal dies quietly, without dyspnœa or struggling.

Sapræmia, as it occurs in man, generally results from absorption of considerable quantities of decomposing matters in cases where pus and the other products of inflammation have been retained under tension. Thus, it may be caused by decomposition and retention in the uterus of the fluid discharge which follows parturition, or by the retention of pus between the flaps of a septic stump.

Septicæmia, on the other hand, may be produced in man, as in mice, by a slight prick with any weapon which has

been dipped in septic material, and is particularly prone to follow such injuries as dissection and post-mortem wounds, or the injuries sustained by butchers and others who have to deal with dead animals. The amount of poison introduced may be infinitesimal, but it can increase in the body to an almost indefinite extent.

It is evident that, in the case of sapræmia, if the dose has not been large enough to kill, the patient may readily recover if the cause of the poisoning is removed—*e.g.*, if the retained pus is evacuated. On the other hand, in true septicæmia, even if the cause be removed, the patient is likely to die, as the micro-organisms, once introduced, continue to multiply. A fatal termination is not, however, inevitable, and in some cases at least the bacteria are destroyed by the tissues, and the patient recovers.

The **symptoms** of "septic intoxication" in man cannot practically be separated from those of "septic infection," and it is probable that in many cases the two diseases co-exist. They would never have been differentiated except by experiments. The symptoms, and the course they run, can scarcely be considered at length in the present work, but it may be briefly stated that, after an initial rigor of long continuance and unusual severity, which is not repeated, there follows extreme depression of all the vital functions, and most noticeably of those of the central nervous system. All the secretions are dried up, and urine may be completely suppressed. The secretion of pus shares in the general disturbance, and any wound that there may be becomes dry, and is sometimes covered with a yellowish rind. Severe frontal headache is throughout a most prominent symptoms.

The circulation is greatly interfered with, especially in the smaller capillaries, and in many parts of the body congestions and actual stasis result. The blood may also escape from the vessels, and may form small ecchymoses or petechiæ. These may be seen in the skin, but are much more common on mucous and serous surfaces. If a drop

of blood be drawn off during life, microscopical examination shows that some of the red blood-cells are already undergoing disintegration, and that all of them exhibit a great tendency to cohere and form solid masses. The breaking up of the red blood-cells indicates that the poisonous material actually possesses the power of destroying the essential elements of the blood, whilst the cohesion of the cells also explains, in part at least, the tendency to stasis and congestion.

On post-mortem examination, decomposition is found to be far advanced, and rigor mortis frequently but little marked. Incisions into the muscles of the chest and limbs show that the latter are stained by the blood-colouring matter which has escaped from the disintegrated cells. On opening the chest, petechiæ may be discovered on the pleuræ and pericardium. The heart-substance is stained, as are the voluntary muscles, and, like them, it is extremely soft and tears easily. The blood in the heart-cavities and throughout the body is of a dark, tarry colour, and is either fluid or only imperfectly coagulated. The lungs are very congested and lacerable. The peritoneum may present petechial spots, and the liver, spleen, and other viscera are all darkly congested and very friable.

PYÆMIA.

Pyæmia is a constitutional disease due to absorption of a septic poison from injured tissues, and is characterised by the development of numerous secondary metastatic inflammations.

In pyæmia, as in septicæmia, it is probable that definite forms of organisms produce the poisonous or septic matter, the absorption of which into the system gives rise to the general or constitutional symptoms of the disease. In both pyæmia and septicæmia alike, the wound which is the seat of inoculation is generally of recent origin, and wounds which are granulating are rarely infected. It is now believed that the organisms present in pyæmia

are the same as those which produce the poison in septicæmia.

The earliest symptom of pyæmia is a rigor, but, whereas in septicæmia the rigor is not repeated, pyæmia is characterised by frequent rigors. The length of time that pyæmia takes to run its course varies greatly in different cases. In some, the patient dies in three or four days; in others, not for weeks or for months. Even the most rapidly fatal cases survive longer than do those of septicæmia. The most frequent complications are broncho-pneumonia and pleurisy, pericarditis and endocarditis, abscesses scattered throughout the body, painful swellings and suppurations of joints, peritonitis, and thrombosis of the veins in the neighbourhood of the seat of inoculation.

On post-mortem examination the muscles may be found more or less stained as in cases of septicæmia. The lesions most commonly found comprise the following:—On opening the thorax, the pleuræ contain considerable quantities of dark, dirty-brown, foul, blood-stained fluid, often mixed with pus and shreds of fibrin. Similar fluid may distend the pericardium, and the surface of the latter membrane may be rough and shaggy with recent lymph.

On removing and washing the lungs, small raised patches, varying in size from a pea to a walnut—and seldom larger—may be seen close beneath the pleural surface. The smallest of these are dark in colour, the largest opaque white. If the former are incised, they are found to consist of very darkly congested patches of lung-tissue, into which a small amount of blood has been extravasated. If one a little bigger be chosen for incision, it will be seen that in the centre of the darkly congested area there is a drop or two of pus, whilst all the swellings larger than a hazelnut are nothing more than abscesses. On making sections of the lung, patches of broncho-pneumonia will usually be found, and there may be pus in the smaller tubes. No abscesses, however, are usually found deep in the lung itself; they are confined to the sub-pleural surface.

A microscopical examination shows yet more, for the vessels in the thrombosed area are found to contain numerous micrococci, which in some of the smaller capillaries are in such numbers that they alone are sufficient to arrest the passage of blood (see Fig. 7).

On opening the heart, endocardial inflammation or small abscesses in the heart-muscle may be found. The peritoneum may contain fluid similar to that met with in the pleural cavities and pericardium, but its occurrence is not nearly so frequent as in the latter situations. The spleen is large, soft, and friable; the liver is in a similar state.

Fig. 7.

A Small Vessel in the Lung completely plugged with Micrococci. From a case of pyæmia. (Crouch, $\frac{1}{6}$ in.)

Either viscus, as well as the kidneys, may contain abscesses. If one of the joints which has been swollen and painful is laid open, it is found to be distended with thin, oily pus. Frequently the cartilages and synovial membrane look quite healthy and shiny; in other and rarer cases they are ulcerated, and the bone is exposed. Abscesses may be found in different parts of the body, being specially common in the parotid regions. Wherever pus occurs it contains micrococci.

An examination of the wound will generally reveal the presence of retained pus, which is often foul. The veins in the neighbourhood are sometimes filled with clot, and thrombosis may extend for a considerable distance. The clot is always ill-formed, friable, and disintegrating. Frequently, in parts which are the most broken down, either

semi-purulent fluid or true pus may be found. The vein-wall may be roughened or ulcerated.

EXPLANATION OF THE SECONDARY ABSCESSES.

Such are, briefly, the post-mortem appearances of a case of pyæmia. Some of them—*e.g.*, the inflammation of the synovial surfaces—may be explained by the poisoned condition of the blood, but it is evident that the abscesses in the different viscera require further explanation. The first thing to notice about them is their locality. They are far more frequently met with in the lungs than in other viscera, and, whenever they occur, are situated at the periphery of organs and in the neighbourhood of the smallest capillaries. This at once suggests an embolic origin, and the source of embolism is readily found in the thrombosed veins. If an embolus derived from one of the valves of the heart, or from a healthy clot, and consisting simply of a piece of fibrin, be lodged in the terminal branches of any visceral artery, it will cause a complete stoppage of the blood-stream, with secondary congestion, but in no case will suppuration ensue. The abscesses of pyæmia cannot, then, be accounted for by the lodgment of simple emboli. No; the *condition of the clot* in the veins furnishes the necessary clue. The wound has been the seat of certain septic changes due to the presence of micro-organisms, and septic matter has been formed in it. Similar changes have occurred in the veins and their contents, and it must be remembered that the coagulated blood is dead tissue, and therefore incapable of offering any resistance to the growth of the parasites, which indeed it supplies with food. The clot is not a simple thrombus; it is a poisoned or infected one, and contains micrococci, and because it is so poisoned, therefore it disintegrates. The abscesses now are easily explained. The clot in the vein breaks up, and minute portions of it, being washed away, are carried by the venous blood to the right side of the heart, and are thence propelled into the pulmonary

circulation. In the smallest arterioles these emboli lodge, and in the vessels and the neighbouring lung-tissue the micrococci excite exactly the same septic changes as were occurring in the wound from which they have been directly derived. The emboli are not simple; they are *infective*.

The abscesses in the other viscera are similarly accounted for. Either some of the particles of clot from the original seat of thrombosis are not detained in the lung-capillaries, and, reaching the left side of the heart, enter the systemic circulation, or, as is much more probable, fresh bacterial emboli are derived from disintegrating clots formed in the pulmonary veins around the scattered abscesses. The embolic nature of the abscesses in question is further exemplified by noting that, in cases of pyæmia following injuries or operations about the rectum, the abscesses are most frequent in the liver, and not in the lungs—a fact which is readily explained when it is remembered that much of the rectal blood enters the portal and not the systemic system of veins. In "ulcerative endocarditis" also, where the valves of the heart are the seat of bacterial growth, the emboli which are frequently detached lodge in the arterioles of the spleen, kidneys, brain, and other viscera, and cause the formation of embolic abscesses, whilst at the same time septicæmia results from the general poisoning of the blood.

It will thus be seen that, in pyæmia, there is not only septic fluid absorbed from the wound, but solid particles of poisoned clot are scattered by means of the circulation, and it is these solid particles which give rise to the metastatic suppurations which are typical of the pyæmic state. But the abscesses in the lungs and heart are also the cause of the suppurative pleurisy and pericarditis already described. As has been mentioned, these abscesses are situated just beneath the serous coat, and as they increase in size they commonly rupture into the serous cavity. The septic pus they contain being thus brought into contact with the pleura or pericardium, suppurative inflammation of these membranes ensues.

Pyæmia is really merely a variety of septicæmia, characterised by the presence of secondary or metastatic abscesses, but produced by the same organisms. The process is essentially septicæmic, but there is a formation of infective thrombi, which necrose and lead to embolism, and hence to fresh metastatic foci, which suppurate and act as additional "septic" and intoxicative centres. The symptoms are in the first place due to intoxication, but in part also to the mechanical effects of embolism.

It will be seen, then, that in a case of septic infection, when perchance the organisms obtain access into the circulation, one or more results may ensue: (*a*) they may grow and multiply in the blood, causing generally rapid death by means of an acute blood intoxication: (*b*) the blood stream may deposit the organisms throughout the tissues, and thus cause a general diffusion of the infective process, as, *e.g.*, in cases of ulcerative endocarditis resulting from a septic infection where occasionally a general erysipelas of the face and extremities may be produced through the circulation; (*c*) thrombi may form at the seat of injury, which become infected and break down; the detached clot then plugs some distal vessel, and embolism results, leading to secondary septic foci (pyæmia). If the blood be examined during life, micro-organisms are invariably found in the first two conditions, and are rarely absent in true embolic pyæmia. These are the essential differences between the various modes of infection through the circulation. It is difficult to find special names for all the various "septic" processes, but pyæmia as a clinical term can be applied only to a septicæmia accompanied by metastatic infective embolism.

No doubt typical instances of these conditions differ both clinically and anatomically from each other, but frequently no hard-and-fast line can be drawn. Thus, in typical septicæmia there is but one rigor, and death ensues very rapidly; but in other cases there are several rigors, and the patient may live a week or ten days, yet a post-

mortem examination shows the lesions of septicæmia alone, and none of the multiple abscesses of pyæmia. Again, a case which, at first at any rate, looks like one of septicæmia may exhibit after death numerous disseminated suppurations. It is on account of such cases and of bacteriological observation that we must consider these diseases as really and essentially parts of the same processes, and that the entrance of the organisms into the circulation, and the disintegration of septic thrombi are to be considered as chance occurrences in the course of a case which otherwise would be called simple septicæmia.

The conditions of wounds and of patients which predispose to the development of the diseases in question are identical. Overcrowding, insufficiency of fresh air and good food, the presence of large numbers of suppurating wounds in the same room or building, are the most important. Wounds of veins or of bones are more frequently followed by both pyæmia and septicæmia than injuries of other parts of the body; whilst the retention of decomposing pus and other inflammatory products is always a fertile source of such affections.

The following table may assist in giving a clearer idea of the various processes of septic infection.

INTOXICATION.

A. Following an infection by *saprophytic* organisms = Sapræmia.

B. Following an infection by *parasitic* (*pyogenic*) organisms. = Septicæmia.
- (*a*) Spreading along the lymph channels.
- (*b*) Spreading by means of the blood-vessels:
  - (1) Growing and multiplying in the blood.
  - (2) Carried to the tissues by the blood.

C. Following an infection by *parasitic* (*pyogenic*) organisms. = Pyæmia.

Septicæmia with infective thrombi and emboli.

CHAPTER IX.

ERYSIPELAS.

Erysipelas is essentially a spreading infective inflammation. It usually attacks the skin, but may also occur on mucous, serous, and synovial surfaces. It is probable that it always commences in a wound, for cases of so-called idiopathic erysipelas affect almost invariably some exposed part of the body, such as the face, where small abrasions are common. The disease is the result of a local infection, but although the chief apparent effect of the inoculation is also local, there is always at the same time a more or less severe constitutional disturbance. It will be convenient to describe erysipelas as occurring on cutaneous surfaces, for here it is at once most frequently met with, and is seen in its most typical forms.

Three varieties of the disease are described:

Cutaneous erysipelas.

Cellulo-cutaneous or **phlegmonous erysipelas.**

Cellulitis.

In each of these the first symptom is a rigor, with rise of temperature, followed by sweating. Any existing wound assumes an unhealthy aspect, healing processes cease, granulations wither, and secretion of pus diminishes. Very soon the edges of the wound swell, and the skin becomes bright red. The swelling and redness extend together, so that the limits of a **cutaneous erysipelas** while it is spreading are sharply defined by a raised, well-marked border. The hyperæmia is followed by exudation of fluid, which collects beneath the epidermis to form vesicles, or else, accumulating

in larger quantities, results in the production of bullæ. The exudation is not limited to the skin, but extends also to the subcutaneous tissue, and when the latter is loose, as in the eyelids and scrotum, the effused fluid may cause much swelling. Very shortly after the first symptoms of erysipelas, the lymphatic glands in the neighbourhood become inflamed and swollen, and soon afterwards the lymphatics between them and the wound are marked out as tender and red lines. As the erysipelas spreads at the edges, it fades in the centre, and, when it ceases to spread, the raised, defined margin is lost, and the redness fades gradually into the surrounding tissues. It is not usual for cutaneous erysipelas to terminate in suppuration, but this may occur. After the redness has begun to fade, the epidermis of the affected area quickly desquamates. The symptoms of fever are always well marked, but the amount of constitutional disturbance depends greatly on the condition of the patient's health.

In **phlegmonous erysipelas** the inflammation is not confined to the true skin, but extends more deeply, and involves the subcutaneous tissues. The redness of the skin is not so bright as in the cutaneous form, but the swelling is greater. The exudation contains much more of the fibrin-forming elements of the blood, and as a consequence causes great induration and brawny thickening; the inflammation is always very acute, and exudation most rapid. On account of the rapidity with which exudation takes place, the inflamed parts soon become very tense and shiny, their vessels are compressed, and they "slough" from interference with their nutrition. Almost all cases of phlegmonous erysipelas result in suppuration, and after a time the brawny thickening gives place to a softer and boggy feel, which indicates the liquefaction of both inflamed tissues and inflammatory products alike. At this stage the epidermis frequently peels, and, especially when no treatment is adopted, large areas of skin die, together with portions of the subjacent areolar tissue. If the erysipelas now subsides, the dead tissue will be

cast off as a slough, and an ulcerating surface will be exposed, In bad cases the sloughing process involves tendons, muscles, and vessels, and may leave a limb which is permanently damaged or useless. Constitutional symptoms are much more marked in phlegmonous than in cutaneous erysipelas. The temperature frequently rises to 105°, or higher, and delirium is common. One of the most common complications is bronchitis with congestion of the lungs, or broncho-pneumonia.

Cellulitis differs but little from phlegmonous erysipelas. In it, however, the stress of the inflammation falls primarily on the cellular tissue, and secondarily on the skin. As a result of this, redness is not so marked, and is usually preceded by swelling. Inflammation of the lymphatic vessels and glands is common, and suppuration and sloughing occur as in the cellulo-cutaneous form.

If a patient dies of any of the varieties of erysipelas, a post-mortem examination frequently reveals but little. The most common morbid condition is great congestion and œdema of the lungs, often combined with broncho-pneumonia and pus in the smaller tubes. Occasionally pleuritic effusion is found, and when the head has been the seat of the inflammation, there may be diffuse meningitis. The abdominal viscera may be congested, and the blood more fluid than natural.

Causes of erysipelas.—It is probable that extreme plethora predisposes to erysipelas, and there can be little doubt that some individuals are, without apparent cause, specially prone to be attacked. The disease appears to be induced by chronic nephritis rather than by affections of the other viscera, and in those fatal cases which I have examined after death I have almost always found catarrhal or interstitial inflammation of the kidneys. The accumulation of large numbers of suppurating wounds in a building, and exposure to bad smells or draughts of cold air, act as exciting causes, whilst the retention of inflammatory products in a wound is always fraught with danger of erysipelas.

Recent experiments have made it certain that specific micro-organisms play the most important part in the causation of this disease, and the undoubted influence of such causes as have just been mentioned in no way tends to disprove the influence of organisms, for it is probable that the latter are only capable of causing trouble when under circumstances that are favourable for their growth and development.

It has been shown that in erysipelas the lymphatics of the skin are filled with micrococci (streptococcus erysipelatos), and the disease has been readily transmitted to other animals by inoculation either of fluid containing these organisms, or else of the micrococci themselves after many cultivations carried on in different media. These experiments have been made on man as well as on animals, and in almost every case typical erysipelas followed inoculation. From the rapid extension of the micrococci along the lymphatics, erysipelas has been called "infective capillary lymphangitis." The streptococcus of erysipelas is closely allied to, if not identical with, the streptococcus pyogenes. Recent authorities consider it a variety of the pyogenic organism, and it would seem that the latter under certain conditions will produce a mere suppuration or abscess; under others, erysipelas; and under others, again, the severest forms of septicæmia. Why this should be so it is as yet impossible to discuss, but probably it depends on susceptibility and predisposing causes.

CHAPTER X.

TETANUS.

Tetanus is a disease which usually follows an injury, and is characterised by tonic and clonic spasms of various muscles.

The injuries which are most frequently followed by tetanus are lacerated wounds of the hands and feet, but it may well be doubted whether this frequency is out of proportion to the exposure, and consequent liability, of such parts to sustain hurts. Tetanus rarely occurs within the first twenty-four hours after injury, and its onset may be postponed for several weeks.

In almost all cases the wounds are contaminated with dust, dirt, or earth. Tetanus may follow the smallest wound or the merest scratch into which such matter has obtained access, for the bacillus of tetanus is found especially in the soil and in the dust. It is therefore widely distributed, but it seems that tetanus is commoner in warm climates, though this is possibly due to the fact that in such countries people walk about less protected. Farmers, gardeners, and labourers are especially exposed to the infection, and tetanus is not rare on battle-fields. The bacillus of tetanus is an obligatory anaërobic organism, capable of forming spores. These spores are terminal, so that the spore-bearing bacillus under high magnification resembles a drumstick (see Fig. 8). It occurs in earth and soil down to a depth of six to seven feet, beyond which bacterial life is impossible, and a small trace of such earth inoculated under the skin of a mouse or guinea-pig will produce typical tetanus in about 12–24 hours.

At one time various theories existed as to the causes of tetanus, but now it has been settled beyond all doubt that it is caused by the specific organism. The latter is found at the seat of lesion in tetanic patients, or can be cultivated from the pus or exudation of the wound, or from the surroundings of the patient. It has been shown that the tetanus of puerperal women and that of new-born children are caused by the same bacillus. In fact, without that organism there can be no tetanus, so that the older views of idiopathic or spontaneous tetanus must now be definitely abandoned. It was assumed that abnormal conditions of the peripheral nerves at the seat of injury, either by directly extending to the spinal cord, or by reflex irritation, were capable of exciting general spasms. This is now known to be without foundation.

It is important to remember that tetanus is essentially an intoxicative lesion and not a true septicæmic process. After death the toxine has been obtained from the blood and various organs, though the urine remains free. The organisms are found apparently only during the early stages of the disease, at the seat of injury alone, and as a rule in such small numbers that their presence cannot be demonstrated except by a timely animal experiment or artificial cultivation in hydrogen, or at any rate in an atmosphere free from oxygen. The bacilli produce an extremely active poison or toxine—·0003 grains is the lethal dose for a man—which is absorbed, and, acting on the spinal cord, excites the typical spasms. The nature of this substance is unknown, but it seems to be an albuminoid body, perhaps allied to the enzymes. It may be obtained from artificial cultures, and injected into a susceptible animal it will readily produce fatal tetanus.

FIG. 8.

Bacilli of Tetanus.

There is a great difference in the resistance of animals to this disease: rodents quickly succumb, dogs are but little predisposed, and hens naturally immune. It is inter-

esting from a clinical point of view that the relative insusceptibility of dogs breaks down, if concurrently with the tetanus bacillus pyogenic organisms be injected into the tissues, for it seems that clinically the prognosis is most serious where the wound is in a condition of suppuration. After artificial inoculation the spasms appear within a few hours, occurring first in the inoculated limb, whence they quickly spread to become general. In man the incubation period varies from one day to two weeks or longer. The shorter the interval is, the worse the issue will be, and cases of an incubation period of from 8–14 days frequently recover without treatment.

The wound very frequently is, or has been, in a sloughy and unhealthy state, but occasionally it is already healed when the symptoms appear, and no trace of an injury is to be detected. Such cases are often, though wrongly, called "idiopathic."

The first complaint is generally that the neck feels stiff, as if from exposure to cold, and the movements of the jaws are soon impaired. After a time, swallowing causes spasm of the muscles of deglutition, and the jaws become clenched. Tonic spasm of other muscles ensues, and the abdominal walls consequently become hard and resistant In many cases the muscles of the back are in a condition of tonic spasm, and the patient is bent backward into a position of opisthotonos. In addition to the tonic contractions, the patient is liable to be seized with sudden and violent clonic spasms, which affect the whole muscular system, and in the case of the diaphragm give rise to severe "girdle" pains. Constipation is common, and retention of urine may occur. The mind remains clear throughout. The act of deglutition becomes impossible after a time, and any attempt to swallow excites spasm. Death may result from exhaustion or cardiac failure, but it is more frequently due to spasmodic closure of the glottis or to prolonged spasm of the muscles of respiration. The temperature frequently presents no material change, but occasionally it rises as high as 107° or 108°, and

in such cases not infrequently continues to ascend after death.

Post-mortem examination rarely reveals any abnormal conditions. The viscera are usually quite healthy, but some of the muscles may be found to have been torn by the violence of their own contractions. The brain and spinal cord are quite normal to the naked eye, and, although inflammation of nerves in the neighbourhood of the wound has been described, it is certainly so rarely seen that its occurrence must be regarded as problematical, and its importance more than doubtful.

Serum or antitoxic treatment of tetanus.—Recently Behring and Kitasato have shown that from animals artificially immunised against tetanus a serum may be obtained which when injected into other susceptible animals of the same or another species will render them also immune—*i.e.*, the serum is protective or immunising. They have further demonstrated that it is possible to cure the disease by means of repeated injections of the immunising serum, but it requires, as may be guessed, much larger quantities of serum for the purposes of a cure than for immunisation. Naturally the efficiency of the serum depends on the degree of immunity established, and to a certain extent also on the species of animal used. It seems that more susceptible animals after immunisation give a serum more active than one obtained from less susceptible ones. The serum of a naturally refractory animal, like the hen, has no protective or curative powers, but if the animal be first inoculated with large doses of tetano-toxine its serum will become potent. The principles underlying these facts are at present not fully understood. The serum possibly contains substances which act either on the tetanic poison—*i.e.*, *antitoxic* substances—or which destroy the organisms—*i.e.*, *antibiotic* or *germicidal* substances.

The protective serum mixed in a test-tube with tetano-toxin will render it inert, or "neutralise" it, so that it actually seems that the action depends on some obscure anti-

toxic property, but it must not be left out of sight that the natural predisposition or resistance of the animal are factors of great importance. It has been shown, for instance, that a dose of toxine just neutralised with serum, so as to be harmless for white mice, is still harmful to the more susceptible guinea-pig. Serum immunity must depend on many obscure changes and conditions in the animal body, and at present we must rest content with the facts.

These animal experiments have been applied to man with some slight success, the curative serum being obtained, as a rule, from rabbits, dogs, or horses which have been highly immunised. It appears, however, that cases of acute tetanus, with an incubation period of from 1–7 days, are hardly ever benefited, 80 per cent. at least dying in spite of the serum treatment; but in less acute cases the serum, or antitoxine as it is called, is of use, and we are already in possession of favourable records, where the mortality amounted to only 11.15 per cent. It is at present too early to form an opinion as to the value of this new method, but the serum treatment seems the only rational mode of attacking this deadly wound infection.

French observers have advised the use of the serum as a protective agent in cases of lacerated wounds or crushes, where particles of sand or earth are rubbed into the sore, or to employ it on the battlefield where tetanus is not uncommon and extremely fatal. The serum is to be injected immediately, so as to remove a possible danger of infection. Immunity is readily produced in this manner, and a small dose only is required.

The serum treatment has recently also been applied to diphtheria after Behring had established its soundness by patient researches on animals. Diphtheria being due to a poison which is less potent or rapid than tetano-toxine is much more amenable to this mode of treatment than acute tetanus, and after a short period the diphtheria mortality has already been considerably reduced.

CHAPTER XI.

GANGRENE.

The term gangrene is equivalent to death, but is generally applied to death of large portions of the body, and not to that of small pieces of skin or cellular tissue. The death of these is spoken of as taking place by the process of "sloughing," and the dead portions of tissue are called "sloughs." In ulcers, as already described, death is "molecular," and the dead particles are too small to be recognisable by the naked eye. Gangrene is usually due to interference with the circulation. If the supply of arterial blood be alone arrested, the tissues will not only die, but, being deprived of all fluid, will rapidly shrivel and dry up. In consequence of this, gangrene resulting from arterial obstruction is usually classified under the head of **dry gangrene**.

If the venous or capillary circulation is also interfered with, the dead parts will retain fluid in varying amount, and, remaining moist and succulent, are said to be in a condition of **moist gangrene.**

In other cases the death of the parts is the result of the action of some poisonous and usually septic matter introduced from without, and hence gangrene from this cause is classified under the head of **septic gangrene.**

In addition to these causes, gangrene may result from the action of some chemical agent such as strong nitric or sulphuric acid or caustic alkali.

DRY GANGRENE.

Dry gangrene is caused, as we have already said, by stoppage of the supply of arterial blood; it may be induced

in various ways. In perfectly healthy people with healthy vessels it may be caused by any **injury to the main arterial trunks**—*e.g.*, subcutaneous laceration, pressure by a fragment of a fractured bone, the application of a ligature, &c., as well as by the **lodgment of an embolus** in some vessel too small to admit of its transit. In a very large number of patients who meet with such accidents the collateral circulation is quite sufficient to supply the peripheral parts with blood, but in others, and especially when the vessels are diseased or plugged by secondary emboli detached from the thrombus found at the original seat of embolism, this is not so, and gangrene results. The causation of gangrene is further favoured by the fact that the embolus commonly lodges at a bifurcation, and so occludes two vessels, as well as by the diseased state of the heart often present as the cause of the embolism. In such cases the patient usually suffers at first from severe pain of a burning character, pulsation ceases in the vessels below the seat of obstruction, and the limb becomes anæsthetic, cold, and pale. The onset of the symptoms is sudden, the pulsation in the occluded vessel can be felt to stop at the point of obstruction, and in this situation there is frequently some tenderness. In most cases the extent of the gangrene declares itself within forty-eight hours, and after this time it ceases to spread. If, however, the other vessels of the limb are diseased, the gangrene is not so rapidly defined.

As the limb dies, the colour changes from a dead white to a bluish tinge, and then, if left long enough, gradually assumes a dull-red, greenish, or blackish hue. The skin shrinks into folds, and the epidermis becomes dry and horny. Finally, the gangrenous part is completely mummified, and in this state may easily be preserved for many years after removal from the body. Such complete mummification is rare, and usually certain parts of the dead limb are succulent and decomposing.

The dryness and blackness of the gangrenous part are

most marked at the periphery, and where the dead skin joins the living the former is of a dull-red colour, and comparatively moist. If the gangrenous part be left in contact with the living tissues, the latter soon show evidence of inflammation. A bright red line appears at the junction of the living with the dead; exudation of inflammatory products proceeds with rapidity, pus is formed, and ulceration commences. In this way the dead parts are separated from the living by what is commonly called the "**line of demarcation.**" This line, as will be seen, is the result of a process of ulceration by which those living tissues which are in contact with the dead parts are destroyed, and so separated from the latter. The ulcerative process, once it has begun, progresses so long as the dead part lies in contact with, and so produces irritation of, the living. In this way a gangrenous portion of a limb may be completely separated and cast off, though such a result will necessarily occupy much time if there is any bone involved. After separation has been completed, the granulating surface which is left will heal like any other ulcer.

Senile gangrene is a variety of dry gangrene dependent upon disease of the peripheral arteries. In old people the vessels are frequently the seat of atheroma, or of primary calcareous degeneration. As a result of either of these morbid conditions, the arterial walls become very tough, rigid, and inelastic, and in proportion as they become so altered they are incapacitated for the proper performance of their functions as carriers of blood. The peripheral parts are consequently not properly nourished, and in some cases the deficiency in the blood-stream is aggravated by the clotting of the blood on the roughened vessel-wall, and the occlusion of the artery by thrombosis. These changes are most commonly met with in the arteries of the lower extremity, and thus senile gangrene occurs most frequently in the foot and leg. In many patients with senile gangrene, the heart also is fatty, and the general health feeble.

The limb of a person whose vessels are thus diseased is usually cold, and is liable to become readily numbed from exposure, or if subjected to compression of any kind; the arteries themselves may be felt as hard, rigid, and tortuous cords. If a toe of this ill-nourished limb be in any way injured, the slightest inflammation may produce an extensive stasis of the feeble blood-stream, and may thus completely deprive the parts of blood. Gangrene of the injured toe will now ensue, and the tissue in contact with it becomes in its turn the seat of inflammation, which again induces stasis and gangrene. The fact is, that the vitality of the tissues is so low that they are unable to withstand the slightest interference with their nutrition, and the gangrene, having once started, will continue to extend till its progress is arrested by meeting with some tissue in which inflammation may progress to ulceration, and to separation of the living from the dead part by the formation of a "line of demarcation."

In some cases the gangrene is of the dry variety throughout, but most frequently the inflammation which precedes it in its course up the limb results in the exudation of a certain amount of fluid, and until this has been dried off, the dead tissues remain moist. The gangrenous part is thus always most dry at its periphery, where it has been longest dead.

In many cases, after a line of demarcation has commenced to form, gangrene again begins to extend, an event which means that the parts reached by the gangrene are not sufficiently well nourished to withstand the call made upon their vital resources by the inflammatory process. Considering, therefore, that the parts about any line of demarcation are, so to say, only just alive, it is at once evident that any additional injury inflicted upon them, such as the cutting of a flap for an amputation, will be the cause of a further extension of the gangrene. The appearance of the dead tissues and the mode of formation of the line of demarcation differ in no material respect

from the similar processes already described in dealing with the subject of gangrene from embolism and from injury.

Patients who are the subjects of senile gangrene are usually in a very feeble state of health, and in many cases the pain, want of sleep, and absorption of septic products from the decomposing tissues cause a fatal termination before the gangrenous process ceases. Death occasionally results from pulmonary embolism.

Diabetic gangrene.—Persons who are suffering from diabetes are liable to be attacked by a form of gangrene which resembles in many respects senile gangrene, although the dead parts do not shrivel and dry as in the latter, but, on the contrary, are often moist and decomposing. Diabetic gangrene is frequently started by some trifling injury, and spreads rather quickly up the affected limb. Its course is more rapid than is that of senile gangrene, and it evinces very little tendency to limit itself by the formation of a line of demarcation. The sole of the foot is the commonest place in which diabetic gangrene commences, and in some cases a "perforating ulcer" is the beginning of the disease. These cases must be clearly separated from another and rather numerous class in which *after* gangrene has commenced as a result of disease in the vessels or from other cause, sugar begins to be excreted with the urine. Here the patient has a transient glycosuria, and the sugar in the urine is the result of the gangrenous process, and in no way associated with its cause.

Idiopathic symmetrical gangrene, or **Raynaud's disease,** are the terms applied to certain cases of dry gangrene occurring without any evident cause, and usually, but not always, affecting parts symmetrically placed.

The patients are generally young adults or children. The parts affected are the fingers and toes, and more rarely other portions which are exposed, such as the ears and the nose. Very commonly there is a history that, for some time previous to the appearance of the gangrene, the

extremities have been numbed and cold, with enfeebled circulation and a tendency to the formation of chilblains. Death commences at the extreme periphery, and extends upwards. One or several digits may die, and more rarely the gangrene extends to the whole hand or foot. In most cases the dead parts are extremely dry and mummified. Prognosis is good as far as life is concerned. The dead parts are separated in the usual way, and surprisingly good stumps are formed. The real cause of the gangrene is yet obscure. It is probably of neurotic origin, and in some cases at least is the result of peripheral neuritis.

Gangrene from frost-bite.—Parts which are frozen for a sufficient length of time do not recover their vitality on the removal of the cause. The result of exposure to extreme cold is a contraction of the arteries, which, if excessive and long-continued, deprives the peripheral structures of their blood-supply to such an extent that they die and shrivel in exactly the same way as when a large vessel is occluded by a ligature or an embolus. If placed in a warmer atmosphere, blood cannot return to the frozen tissues, for the vessels as well as their surroundings are gangrenous. In such cases the dead part is shrivelled and mummified, and is in a condition of typical dry gangrene.

In other cases, where the deprivation of blood has not lasted so long, it has been noticed that if the part is suddenly placed in a warmer atmosphere, and blood is encouraged to return with great rapidity and in large quantities, gangrene will ensue with much greater certainty than if the frozen tissues are only slowly restored to their natural temperature. In such cases the gangrene is of the moist variety, and is preceded by much exudation and other signs of inflammation.

It is probable that the explanation of this variety of gangrene from exposure to cold is to be found in the behaviour of vessels which have been for some time deprived of their blood-stream. It has been shown, in the chapter

on Inflammation, that the result of such deprivation is that, on the re-admission of blood, the phenomena of inflammation, with rapid exudation, immediately ensue. The peripheral parts, already almost dead, cannot survive any further interference, and accordingly die. The moisture of the gangrenous tissues is the result of the exudation which has immediately preceded their death.

Gangrene from ergotism.—A very brief mention of this variety of dry gangrene is all that is necessary in the present work. The effect of ergot-poisoning is a contraction of the small arterioles, which, when long continued, may result in gangrene of the peripheral parts. It is very rarely met with in this country, where rye-bread is but little used as an article of diet.

MOIST GANGRENE.

In moist gangrene, the dead part, instead of drying and becoming mummified, remains moist and succulent. Unlike the parts in dry gangrene, the dead tissues quickly decompose, for moisture is a necessary factor in rapid decomposition, and is here abundantly supplied.

Moist gangrene occurs in its most typical form after ligature of the main artery and vein of a limb, and may also result from complete constriction, or from the extensive laceration and crushing of a limb which often result from accidents of various kinds. Being deprived of their arterial blood, the tissues perish, and, the venous blood being retained and often widely extravasated, the dead structures do not become mummified. The gangrene is limited to the parts below the seat of injury, the skin becomes blue or livid in colour, and the epidermis, being raised in bullæ, subsequently peels, and exposes the deeper parts of the skin. The whole limb becomes cold, pulseless, and swollen, and, as decomposition advances, gases form and give rise to emphysematous crackling. The deeper structures feel sodden and œdematous, their elasticity is lost, and they retain the imprint of a finger for some

minutes. The living tissues above the occluded vessels quickly inflame, ulcerate, and form a line of demarcation, but, on account of the decomposition and the rapidity with which the gangrene spreads, the absorption of septic material and a fatal termination are much more frequent in cases of moist than of dry gangrene.

Gangrene from acute inflammation.—In many cases of injury the deeper parts are extensively torn, although but a slight skin-wound has been inflicted. The result of this is that, if acute inflammation supervenes, the inflammatory products are retained, and by their presence interfere with the circulation in the injured limb. The more rapid the inflammatory exudation, and the more tense the structures beneath which the effusion occurs, the greater is the interference with the circulation, and the greater is the probability that death of the inflamed structures will ensue. The gangrene in such cases is necessarily of the moist variety, and, although it occurs most frequently after injuries such as have been mentioned, it may occur in connection with acute inflammation and extreme tension excited by any cause—*e.g.*, in phlegmonous erysipelas. It is evident that if exit be given to the pent-up fluid by free incisions the cause of the gangrene will be removed.

SEPTIC GANGRENE.

Septic gangrene is quite independent of injuries to the main arteries or veins. It is probably the result of the local absorption of some "infective" or "septic" material formed in a wound.

Acute spreading traumatic gangrene.—This, which is the best known of the varieties of septic gangrene, is especially distinguished from the other forms of gangrene of which we have been speaking by its tendency to spread. Supposing that a heavy cart-wheel has passed over a man's leg, and that all the vessels have been extensively lacerated, if simple moist gangrene results it will certainly be limited to the parts below the seat of injury; in spreading gan-

grene, on the contrary, the parts above are just as likely to suffer as those below.

The patients in whom this form of gangrene occurs are usually adults, and may be either healthy or unhealthy. The gangrene may follow any injury, but is perhaps most commonly met with after compound fractures. It commences within a day or two of the accident, and is first noticed, not at the peripheral parts, as in gangrene due to injury of vessels, but **at the edges of the wound.** Here the skin becomes dark red, and then green or black. The skin in the neighbourhood assumes a dark-red hue, and the whole limb rapidly swells and becomes extremely tense and œdematous from the presence of exuded fluid. Very quickly indeed the exudation is followed by decomposition, the epidermis peels, and the soft tissues beneath become emphysematous from the presence of the gases which result from decomposition, or from the special activity of organisms, as, *e.g.*, the anaërobic bacillus of malignant œdema, or the bacterium coli commune. The gangrene progresses with such rapidity that within forty-eight hours from its commencement it may have spread over the whole limb and extended to the trunk. Unlike other forms of gangrene, it knows no limits, and **no line of demarcation** forms to arrest its progress. Unless the affected limb be early removed by amputation, death ensues with certainty, the patient sinking into an asthenic condition, and dying without any definite post-mortem evidence of general septicæmia. The temperature is never raised to any great extent in typical cases, and may be sub-normal.

Phagedæna and hospital gangrene.—The term phagedæna is applied to a very rapidly extending and destructive form of ulceration, which is now most commonly seen in connection with venereal sores. Hospital gangrene, or "sloughing phagedæna," is so closely allied to phagedæna itself that the two diseases fade into one another; but, as the alternative name for hospital gangrene suggests, there is, in addition to rapidly extending ulcera-

tion or molecular death, more or less sloughing and death of the tissues *en masse*. This form of gangrene, though formerly of common occurrence in crowded hospitals, is now but rarely seen.

Both hospital gangrene and phagedæna occur independently of interference with the blood-stream, and are each the result of the action of some specific poison, which is either formed in, or inoculated upon, a wounded surface. Both forms of disease are contagious, but hospital gangrene much more so than simple phagedæna.

A phagedænic ulcer has the following appearance :—The shape is irregular and ragged; the base is greyish and often sloughy, or covered with a diphtheritic-like membrane; the discharge is scanty and thin; the edges are sharply cut, and show no sign of healing; the skin immediately around is of a dull red hue. The ulcer is liable to extend indefinitely, but does not often advance to the destruction of such a large amount of tissue as is common in the case of hospital gangrene. The specific poison which is the cause, and the ulcerated surface which is inoculated with it, must be destroyed before a healthy action can be expected. Formerly, strong caustics were the only remedies employed, but of late years it has been shown that, in the phagedæna which affects venereal sores, at any rate, long-continued soaking in water produces all the beneficial effects of the caustic treatment.

Noma vulvæ.—This is a form of phagedæna which affects the external genitals of young children, and is usually seen in connection with dirt and ill-health, though frequently following measles. The appearance of the ulcer does not differ materially from that described above as typical of phagedæna. The general health is often seriously affected, and death not uncommonly results.

Cancrum oris.—This is a form of ulceration with sloughing peculiar to childhood, and is usually met with before the age of five. The ulceration commences in the cheek or gums, and more rarely in the sockets of the teeth.

In bad cases large portions of the cheek may be completely destroyed, and, the ulceration extending to the jaws, necrosis of the bones and destruction of the teeth ensue. The disease is very frequently fatal, and septic broncho-pneumonia or septicæmia may complicate it. Cancrum oris is probably the result of the action of bacterial poisons, and the object of treatment is to destroy the contaminated tissues. This is usually done by the aid of strong caustics.

CHAPTER XII.

BOIL, CARBUNCLE, AND MALIGNANT PUSTULE.

BOIL.

A BOIL is a circumscribed inflammation of the skin and cellular tissue, originating in the hair-follicle or sweat glands, and resulting in the death of the more central parts, and the formation of a slough. This sloughing is accompanied by suppuration of the parts immediately around the dead tissue, and the formation of a pustule which, opening on the surface, permits the discharge of the central slough or core.

Boils are the result of a local infection by pyogenic micrococci and are predisposed to by some disordered state of the constitution, such as may be induced either by excessive feeding or by an insufficient amount of nourishment; sudden changes of diet appear to be a common cause. It has been shown by experiments on man, that if various pyogenic cocci, as for instance the staphylococcus pyogenes aureus, or the staphlococcus citreus, are vigorously rubbed into the unbroken cuticle, that after a short time a crop of boils or furuncles, accompanied by a more or less severe inflammation, will result, the extent of which varies within certain limits with the constitutional condition of the individual experimented upon.

Boils are more frequent in men than in women, and are usually situated on the posterior surface of the body; the back of the neck and of the shoulders, and the gluteal regions, are their most common sites. Any local irritation may

determine the situation of a boil in a person whose general health is in a condition favourable for its development.

CARBUNCLE.

A carbuncle is a specific localised inflammation of the skin and cellular tissue. It is characterised by considerable exudation into the inflamed area, with sloughing of the tissues involved, and formation of numerous pustules. The sloughs are separated from the surrounding parts by suppuration, and, when loosened by this process, are cast off. A carbuncle differs from a boil in the following points:—It is usually single, is much more raised from the surface, and of much greater size. It frequently implicates the deep fascia, and discharges pus through numerous apertures.

Carbuncle is predisposed to by a condition either of plethora or debility, and not infrequently complicates diabetes. The patients in whom it occurs are usually men over middle age. Its most common sites are the nape of the neck and the shoulders. It more frequently produces a fatal termination than does a boil, and death may result either from general asthenia or from absorption of septic material and blood-poisoning.

Malignant facial carbuncle occurs, as its name implies, on the face. It affects people of all ages, but most frequently young adults, and is of decidedly rare occurrence.

Its most common site is the lip, where it commences as a pimple and thence extends to the cheek. Its structure is the same as that of carbuncle in other situations, but it is occasionally associated with a wide-spread thrombosis and suppurative phlebitis of the facial and ophthalmic veins, and with a general cellulitis of the face and orbits. In consequence, the whole face becomes greatly swollen, of a dusky red or purple colour, and the eyeballs may protrude.

The frequent termination of a facial carbuncle is death, and this is most commonly due to pyæmia. It is probable

that in former years some cases at least of "malignant pustule" have been mistaken for facial carbuncle. The most important organism found in carbuncle is the streptococcus pyogenes, and the relation between erysipelas and malignant facial carbuncle is a very close one. When pyæmia or septicæmia supervenes, the streptococcus can generally be obtained from the blood by means of artificial cultivation.

MALIGNANT PUSTULE, CHARBON, OR ANTHRAX.

Certain herbivorous animals—namely, cattle, sheep and horses—are liable to suffer from a disease which is variously known as "splenic fever," "joint murrain," "black quarter," &c., and is caused by the presence of a micro-organism called the "bacillus anthracis." The bacilli anthracis are found in the blood and the viscera of the animal after death, and occur as comparatively long rods, which are slightly swollen at the ends. The extremities are concave and hollowed out, so that a chain of them somewhat resembles a bamboo rod with its nodes. In the animal body spores are never formed, but when grown in artificial media they readily appear under favourable conditions. In broth the bacilli develop into filaments containing highly refractive central endospores, which give the filaments a beaded appearance. The spores are very difficult to kill. An experienced eye can recognise the bacilli easily from their morphological characters, the thickening at the ends and the clear space between two links of the filaments being diagnostic. Mice, rabbits and guinea-pigs are highly susceptible to the infection and the minutest quantity of a virulent culture or a drop of blood from a diseased animal, inoculated subcutaneously, will cause a fatal anthrax septicæmia without fail, the animal often dying within twenty-four hours. Cattle and horses are also easily infected, and epidemics in herds are not at all uncommon in certain countries and districts. Pigeons, hens, and rats are almost completely refractory, but their

resistance may be broken down in many ways, amongst which may be mentioned hunger, fatigue, cold, unsuitable feeding, and enforced thirst. Man also is fairly resistant, and generally suffers from a local affection only, the "malignant pustule." After death, inoculated or infected animals show the presence of bacilli in all capillaries, especially in those of the liver, spleen, kidneys, and lungs, so that a diagnosis is readily made by means of coverglass-films of the splenic blood stained with methylene blue. (See figs. 4 and 9.)

The mode of infection of diseased animals is yet doubtful; it is probable that the organisms can enter either by the alimentary tract or else through wounds on the surface caused by bites of insects, &c. Splenic fever is especially common in Persia and South America.

FIG. 9.

Anthrax Bacilli and red blood-cells.
From Flügge's "Hygiene."

Malignant pustule is a disease produced in man by inoculation with the bacillus anthracis, and is generally due to contamination of some slight excoriation by contact with the hides, wool, or hair of diseased animals. It is consequently most commonly seen on the face, neck, or hands of wool-sorters, tanners, and others who are in the habit of handling portions of infected animals.

Malignant pustule commences as a small, red, irritable pimple. After twelve to twenty hours a vesicle forms and bursts, exposing the deeper layers of the skin. Around this central spot a ring of vesicles now forms, and the contiguous skin quickly assumes a dull-red hue. The central spot dries and becomes black and shrivelled, the tissues outside the ring of vesicles swell, and become indurated and brawny, the vesicles burst and expose more black and shrivelled skin, and the central slough thus increases in size, whilst fresh crops of vesicles are produced, The neighbouring lymphatic glands are often swollen.

Symptoms of constitutional affection are frequently severe, and death is a common result; on post-mortem examination, the tissues present appearances such as are found in cases of septicæmia. Occasionally spontaneous recovery occurs. An examination of the fluid in the vesicles will reveal the presence of numerous pathogenic bacilli, and the latter have also been met with in the sputum, urine, sweat, and blood.

The most typical points about a malignant pustule are the central dry slough, the ring of vesicles, the raised, brawny induration, the absence of severe pain, and the total absence of suppuration.

Occasionally, however, all these conditions may be found in the absence of anthrax bacilli, and may even be so closely simulated by a vaccinia pustule which has become inflamed, that without experimental inoculations the diagnosis cannot be at once established.

CHAPTER XIII.

GLANDERS, ACTINOMYCOSIS, AND LEPROSY.

GLANDERS.

Glanders is a contagious disease, to which horses and asses are subject, which may be communicated to man, and is probably conveyed by means of the secretions of the nostrils. The disease has been proved by Löffler and Schütz to be caused by a specific bacillus—the bacillus mallei. Farcy is simply glanders in a more chronic form.

The lesions of glanders in man are of wide distribution, and occur in the skin, mucous membranes of the nose and pharynx, and the viscera. In the skin the disease is characterised by the development of vesicular, papular, nodular, and pustular eruptions, resembling those of pemphigus and variola, and followed by the formation of irregular and spreading ulcers. In addition to these, nodular masses of various size form on the skin and subcutaneous tissues, and when large and chronic are known by the name of "farcy buds." They are composed of granulation tissue, and after a short time burst, discharge pus, and cause the formation of deep ulcers. In connection with the skin eruption and the farcy buds there is an inflammation of the lymphatic vessels and glands, and subsequently the development of buboes.

The mucous membranes of the nose, frontal sinuses, throat, tonsils, and pharynx, become very swollen and red, and soon covered by numerous small nodules the size of a

pea; these break down and form ulcers, which rapidly extend. In this way the whole of the mucous surfaces in question may become the seat of a most foul and almost gangrenous ulceration.

After death, nodules in various stages of breaking down and suppuration may be found in the viscera, and are of most common occurrence in the lungs. Glanders is a

FIG. 10.

Portion of ulcerated mucous membrane from the nose of a glandered horse. The small circular ulcers at the edge of the diseased membrane are well shown.

disease which is commonly fatal, but the duration of life varies much in individual cases, some patients surviving three or four weeks, whilst others succumb in as many days. A few patients recover.

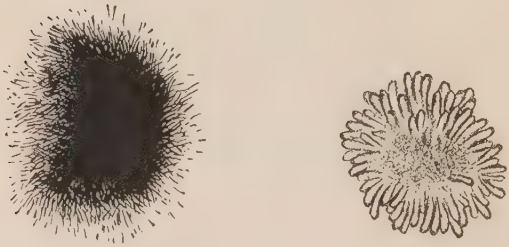
The bacilli are found in all the nodules or ulcers, from which they may be readily cultivated on artificial media, especially on blood serum or potatoes at the temperature of the body. On potatoes their growth is characteristic,

appearing as a yellow or brownish film. The bacilli are a little longer and thicker than tubercle bacilli, and stain with some difficulty with ordinary aniline dyes. They form spores, and resist drying for several weeks. Glanders may be reproduced in susceptible animals, such as guinea-pigs and field mice, by means of artificial inoculations. Recently the toxic bodies have been separated from both cultures (malleïne), and these when injected into diseased horses or asses will produce a marked febrile reaction in most cases, so that they are now employed on the Continent as a means of diagnosis where the disease is suspected but hidden.

ACTINOMYCOSIS.

Actinomycosis is the name given to the diseased condition which follows the introduction into the body of a

FIG. 11.



a. Mycelial network. *b*. Rays and clubs.

The Actinomyces Fungus. From Baumgarten's "Bacteriology."

fungus named the Actinomyces, which probably belongs to the genus streptothrix. This parasite, whose true botanical position, however, has not yet been determined, occurs as minute yellowish-green, brown, or white granules, about half the size of a mustard-seed.

On slight pressure the granules break up into their

several component fungi. The latter probably belong to the group of streptothrix, of which several pathogenic varieties are now known, notably the fungus of mycetoma or Madura disease, to which allusion will be made later. It is star-shaped or crescentic, and consists in typical instances of a central mycelium of closely interwoven filaments and a fringe of hyaline or refractive rays. These filaments are fine and delicate, and in most cases disappear into the characteristic swollen and bulbous or club-shaped rays, which are highly refractive, and stain like hyaline matter with eosine, while the mycelial filaments take up the ordinary bacterial dyes. It must be remembered that the important character of the actinomyces, as of all forms of streptothrix, is the filamentous fungoid mass; the clubs or rays may occasionally be absent. It is still doubtful whether the latter are reproductive or vegetal elements, or merely some hyaline material formed by and around the peripheral threads, for in an artificial growth on the *ordinary* media they do not appear, but only in the animal body, and even then not constantly. Typical representations of the actinomyces will be found in Fig. 11. The fungus can be readily grown on the various nutritive media, and the lesion has been reproduced in animals by artificial inoculation, so that its specificity is established.

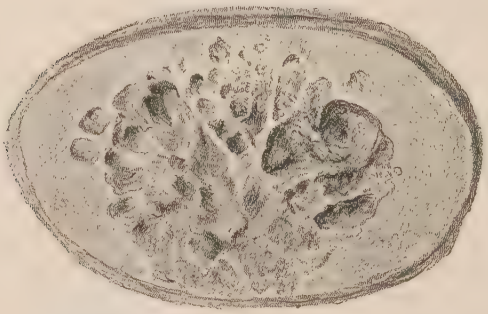
The growth of the actinomyces in the body is always followed by inflammation of the tissue in which it lies, and the consequent production of granulation tissue. In some cases the inflammation progresses to suppuration, abscesses form and discharge, and a fungous swelling of granulation tissue and parasitic growth protrudes through the skin; in other cases, nodules or bands of fibrous tissue are formed, which enclose the fungus, and form definite and considerable tumours. In cases of long standing, the fungus may die and become the seat of the deposit of calcareous salts.

Actinomycosis has long been known to veterinary surgeons as of tolerably common occurrence in cattle and

pigs, and has been very variously named, the most common appellations being "woody tongue," "osteosarcoma," and "lingual tuberculosis." The real pathology of the disease was, however, first discovered by Ponfick in 1877, and Israel was the first observer who described it in man.

It is probable that the parasite grows naturally on barley or corn, and this explains the relative frequency of the

FIG. 12.



Actinomycotic growth inside the lower jaw of a cow. The bone is expanded and hollowed out into numerous cavities which contained purulent matter and actinomycotic growth.

disease in cattle and in rural districts. In more than one case, also, particles of barley, or a whole ear of corn, have been found at the seat of infection. It is not known how the parasite is introduced into the body, but infection of man from animals has not yet been demonstrated. Only a few examples of this disease in human beings have been observed in England. According to Israel, the disease may be transmitted, first, through the mouth and pharynx; secondly, through the lungs; thirdly, through the intestine; but it also occurs in the skin, and has been found in the bladder.

In the first group of cases the fungus appears to obtain

an entrance at a carious tooth, and to extend thence into the jawbone; in other cases the attack has been preceded by pharyngitis or tonsilitis. In the case of the lower jaw, the entrance of the parasite is followed by the formation of a tumour in the maxilla, which expands the bone and extends thence into the submaxillary region. Here in some cases an abscess next forms, and, on bursting, discharges not only pus, but small golden-yellow bodies the size of a hemp-seed, which consist of masses of the fungus peculiar to the disease. The bursting of the abscess does not, however, terminate the case, for fresh swellings may form in the neck, and may extend into the thorax, or into the mediastinum.

In other cases, suppuration does not occur, and a firm, fibrous sort of tumour forms beneath the jawbone. This tumour is remarkable more especially for its change of position, for although formed originally in the inferior maxilla itself, it may gradually sink farther and farther down the neck, leaving at most a fibrous cord to mark the path of its descent. It thus happens that an actinomycotic tumour may, when first seen by a surgeon, be found a considerable distance from the jaw.

In all cases the course of an actinomycotic growth is slow, and there is usually neither pain nor other evidence of acute inflammation. The pus formed is thin and serous, and contains the golden-yellow granules already mentioned. In some cases the discharge is more watery than purulent, and the fluid is copious and viscid. When the growth extends into the upper jaw it may extend to the cheek, or may pass upward, and involve the base of the skull or the vertebræ.

In the second group of cases the fungus appears to be introduced during the act of respiration. It may then localise itself in the bronchi or the lungs, and extend thence to the pleuræ, and so make its way to the cutaneous surface. In some cases the tumours have extended into the pericardium and the abdominal cavity.

In the third group the fungus obtains access to the body through the alimentary tract, and results in the formation of small nodules the size of a pea, or a little larger, in the mucous membrane of the intestine, any part of which may be attacked. These nodules subsequently soften or break down, forming ulcers with undermined edges. From the intestine the fungus may be carried by branches of the portal vein to the liver, and may there cause the formation of large masses of white, tumour-like, soft and honeycombed

FIG. 13.

Actinomycotic growth in human liver, showing the cavities in the liver in which the fungus lay.

tissue, containing the characteristic yellow granules, and arranged in a typically radiate manner (see Fig. 13).

When the parasite has once obtained access into the tissues, it spreads generally along the lymph channels or by continuity. The glands often swell, but this is due as a rule to secondary irritation produced by inflammation or suppuration caused by pyogenic organisms which generally accompany the actinomyces. In rare cases the fungus enters the blood-stream, and a general dissemination results, pyæmic

in its outward appearance, with actinomycotic masses resembling infarcts in the spleen and kidneys, and a general broncho-pneumonia in the lungs, large soft masses in the liver, and even secondary abscesses in the joints and muscles. Almost always the process is a more or less local one, spreading slowly, resembling herein tuberculosis, for which in former years it has often been mistaken.

MYCETOMA OR MADURA DISEASE.

This is a chronic inflammatory affection, which usually attacks the feet, and, more rarely, the hands, and occurs

FIG. 14.

Section of a "Madura Foot," showing the cavities in the bones and soft tissues and the general swelling of the whole foot.

especially in Southern and Western India and North Africa, although isolated instances have been observed in other countries. It is due to a parasite, and affects almost exclusively the native races.

Mycetoma was first recognised as a special disease by Vandyke Carter. It manifests itself in two forms—(1) the black, and (2) the yellow variety. It is characterised by considerable swelling and distortion of the hand or foot, with numerous, somewhat mammillated suppurating apertures, communicating with cavities of various sizes and channels

of various lengths in the subjacent tissues. In the black variety the fluid which oozes from the apertures contains brownish-black particles, in appearance not unlike the rougher description of gunpowder; whereas in the yellow variety, little particles, light in colour, bearing a resemblance to fish-roe, occur. On section in either case, numerous cavities are seen, communicating with each other by sinuous channels, and the carpal or tarsal bones, and sometimes the long bones, present all the appearance of extensive caries, being softened and excavated over large areas. The spaces in the bones and soft tissues alike contain hard, dark masses in the black variety, and a softer, yellowish, gelatinous substance, mixed with globular, roe-like particles in the other. On applying pressure, small quantities of purulent or of oily pus can be squeezed out, together with the characteristic light and dark granules.

As far as naked-eye appearances go, there is an evident similarity between actinomyces affecting bony structures and mycetoma, and recently it has been shown that the yellow fish-roe masses are undoubtedly a form of streptothrix —*i.e.*, a fungus belonging to the same group as the actinomyces. This was proved on morphological grounds by Kanthack, and since then others have succeeded in separating the fungus by cultivation. The yellow variety of mycetoma is hence proved to be due to a vegetable parasite resembling the ray fungus, though not identical with it, but the exact nature of the black fungus is still obscure, since some consider it to be a pigmented or degenerated form or species of the yellow streptothrix, and others are inclined to regard it as belonging to quite a different group of fungi.

The yellow granules consist of a number of individual fungi, forming masses or conglomerations of about the size of a hempseed, often mulberry-shaped, soapy or doughy in consistence. Microscopically, we find a close central mycelium, and generally a peripheral fringe of glassy transparent rays, either clubbed or wedge-shaped, and varying much in

size. In the youngest forms the clubs or rays are absent, but they appear with the progress of the morbid changes. The mycelial threads can be observed to pass into the hyaline rays. The central mycelium is made up of very fine, closely interlacing and interwoven filaments, and presents a close resemblance to the more familiar ray fungus.

The presence of the parasite in the tissues causes first a simple reactive inflammation; soon granulation tissue

FIG. 15.

Madura Foot, showing the general swelling and the lumpy outgrowths.

appears with epithelioid cells and new vessels, and often there is a pigmented zone around the nodule. Gradually the granulation cells are replaced by fibrous tissue, and the result is a small abscess-like mass, or a so-called granuloma, resembling the actinomycotic lesions.

There is no tendency to spontaneous recovery, but, on the other hand, a steadily extending destruction of the affected part, the disease progressing to the complete disintegration of the tissues, and involving the leg or forearm according to the seat of origin. Death results from the prolonged suppuration and its accompanying fever.

With regard to the etiology of the disease little is known, and it is assumed that the fungus finds its way into the human body from the ground or crops. It has not, so far, been observed in cattle. The fungus does not disseminate or spread by the lymphatic or vascular channels, and metastatic growths never occur, nor has the disease yet been found in the internal organs. It is extremely chronic, and radical surgical treatment by amputation as a rule leads to recovery.

LEPROSY.

Leprosy is a constitutional and infective disease, due to a specific bacillus. It is apparently not highly contagious —no more, for example, than tuberculosis—and attacks only predisposed individuals. The predisposition may be caused by bad hygienic conditions, poverty, starvation, climate, &c. The consumption of fish was thought at one time to be the chief exciting or predisposing cause, but there is no evidence in support of this assumption.

Leprosy is very rarely met with in England, but is common in Norway, Iceland, parts of Spain, and in the Baltic provinces of Russia. It is endemic in many parts of Asia, in South Africa, and in Central and South America. It attacks especially in these latter countries the native inhabitants, and although formerly said to be hereditary, it is now proved beyond doubt that the disease is not transmitted from the parent to the offspring. Leprous women, moreover, are commonly sterile.

There are two chief varieties of leprosy—the tuberculated and the non-tuberculated, but each owns the same specific cause and the two varieties are occasionally met with in a single individual.

Tuberculated Leprosy commences often with sensations of chilliness, marked drowsiness and depression, accompanied by occasional profuse perspirations, diarrhœa, and vertigo, and followed by a variable amount of pyrexia. These symptoms are followed by an eruption of pimples or red spots,

which may come out in fresh crops for several weeks or months.

This erythematous eruption is soon followed by the development of tubercles, which vary in size from a pea to a walnut, and are most common and abundant on the face, limbs, scrotum, and penis. They consist of localised infiltrations of the derma with leucocytes and with large round cells about five times the size of a white blood corpuscle, which contain bacilli, and constitute the so-called "lepra cells" of Virchow; the bacilli also occur in the lymph spaces and in the tissues outside the cells. In the growth of the tubercle the sweat and sebaceous glands and the hair follicles are destroyed, the papillæ are flattened out, and the tubercle remains for a time covered by the stretched and thinned epidermis. The further course of the tubercles differs in different cases, and in different tubercles in the same patient. Sometimes the growths shrink and atrophy, leaving depressed cicatrices, but more often many break down and suppurate, forming ulcers which are very slow to heal, and emit a peculiar odour. The tubercles do not affect the scalp, and consequently there is no loss of hair in this situation, but in other parts the hair follicles are destroyed. The nails also break away and become peg-like and stunted. As a result of the development of tubercles, and the thickening of the skin and subcutaneous tissue, the expression of the face is greatly altered; the lips become thick and pouting, the ears are large and pendulous, and the thickening of the skin of the brows and forehead produces an appearance commonly described as "leonine." Tubercles also develop on the mucous membranes, and are relatively common on the throat and pharynx.

Non-tuberculated leprosy is more common than the tuberculated variety; it is also called "**Anæsthetic,**" on account of the loss of sensation in certain parts of the skin. It commences with shooting pains in the course of the nerves, followed by hyperæsthesia and localised pain and tenderness, all of which symptoms are most common in the

extremities. About a year after the commencement of these symptoms a yellowish eruption appears in the form of irregular spots or patches, whose most frequent sites are the shoulders, arms, and elbows, the thighs, knees, and the face.

FIG. 16.

Hand from a case of Leprosy, showing ulceration and clubbing of the tips of the index and middle fingers, and wasting of the muscles, with clawing of the hand.

These patches are characterised by their dryness, all secretion of sweat being arrested, but they are seldom painful or tender. After a time the edges of the patches become raised, and the patches themselves increase in size. Whilst, however, they are thus spreading at their margins they fade in their more central parts, and the skin in these

latter positions gradually assumes a dead white colour like scar tissue, and becomes more or less completely anæsthetic.

The chief symptoms are from this time referable to implication of the nerve trunks in the manner to be presently described. The skin becomes red and glossy, and is occasionally attacked by bullous eruptions; perforating ulcers of the feet, and paralysis of muscles ensue; ulcers develop on the tips of the fingers and toes; the hands and feet become clawed and deformed, dry gangrene or complete absorption of the toes and fingers is common, and thus the hands and feet become maimed almost beyond recognition.

Both the tuberculated and non-tuberculated varieties usually end fatally, from exhaustion due to extensive ulceration, from the cachexia which accompanies the disease, from lardaceous infiltration of the viscera, or from extensive implication of the respiratory mucous membranes.

At a post-mortem examination, in addition to the conditions of skin already described, there is found a general increase of connective tissue in many parts—*e.g.*, in the peripheral nerves, the testes, the lymphatic glands, the liver and the spleen. The growth in the nerves is deserving of special attention, and is most common in the anæsthetic form of leprosy. There can, indeed, be no doubt that the anæsthesia, wasting of muscles, paralysis, ulcerations, and the gangrene of the fingers and toes are mainly caused by constriction of the nerve fibres by fibrous tissue, which also causes considerable thickening of the nerve trunks.

Microscopical examination of the affected parts shows everywhere a definite bacillus, which has been demonstrated in all the tissues above-mentioned as well as in the tubercles in the skin and mucous membranes, and in the cutaneous eruptions, but has not been demonstrated in the viscera, excepting perhaps the lungs. The leprosy bacillus is readily stained in the same manner as the tubercle bacillus but is, however, much larger than the latter, and in length measures about one-half the diameter of a human red-blood

corpuscle. These bacilli are generally held to be the actual cause of the disease, and are supposed, by the irritation which they induce, to promote the growth of the connective tissue which characterises leprosy throughout the body. Inoculations of animals have so far failed to produce this disease.

CHAPTER XIV.

TUBERCLE AND SCROFULA.

Both tubercle and scrofula are more fitly dealt with at length in works on general pathology than in one devoted solely to the surgical aspect of this subject. It is not proposed, therefore, to do more than indicate as briefly as possible the general meaning which will attach to these words when employed in future chapters.

A tubercle, like the lesions of actinomycosis, leprosy, etc., is an infective granuloma of inflammatory formation, and, according to the belief of most authorities, is dependent for its origin on the presence of a micro-organism—the tubercle bacillus. In its most typical form a tubercle appears to the naked eye as a minute, grey, semi-transparent granule, the size of a mustard seed—a "grey tubercle." As its size increases, the more central part of this granule becomes opaque, and is gradually transformed into a soft, fatty, or cheesy mass—a "caseous tubercle."

Microscopical examination of a grey tubercle shows that it consists of a collection of small clusters of cells, each cluster being complete in itself, and forming a so-called "elementary tubercle" or "primitive tubercle." The typical structure of each of these latter is—first: one or more central giant-cells about $\frac{1}{150}$ of an inch in diameter, with a granular protoplasmic stroma containing numerous nuclei, which show a tendency to be arranged in a ring around the circumference of the cell; the cell-body generally sends out processes which may join those from other cells in the neighbourhood. Second: around the giant-cell

is a zone occupied by cells which are larger than leucocytes and have a clear cell-body; they are of connective-tissue origin, but from their likeness to epithelial cells have been named "epithelioid." Third: the outer margin of this zone fades imperceptibly into more or less dense masses of round cells and leucocytes, which in all cases form the outer zone of the primitive tubercle, and in many instances

FIG. 17.

Section of Tubercular Synovial Membrane, showing two giant-cells with numerous processes, and containing many nuclei. (Crouch, 1–6 in.)

make up the great bulk of the whole growth. All the cells throughout the primitive tubercle are embedded in a reticulum of connective-tissue. The whole tubercle is practically non-vascular, blood-vessels not penetrating beyond its extreme margins. The tubercle **bacillus** is from .0015–.0035 mm. in length; it is usually straight, but sometimes slightly curved; it may be found in all parts of a tubercle, but especially in the giant-cells.

Such is the structure of a typical tubercle, but it would be erroneous to suppose that such is the structure of all, for the giant-cells may be absent, or, if present, may not possess

any definite processes, the epithelioid cells may be very few and ill-developed, and the bacillus may not be discoverable. The small-cell growth and the lymphoid reticulum or matrix are, however, constant.

An examination of a tubercle at a later stage shows that the central parts of the elementary tubercles are degenerating so as to form a fatty pulp, a result which is evidently due to the fact that in the growth of a tubercle no new blood-vessels are developed. In some specimens the whole of the growth may be found transformed into a caseous mass.

In any organ affected with tubercle there is a tendency for the disease to spread locally, and to set up in the tissues in which it occurs the processes which are common to all inflammations. On the other hand, an inflamed organ is itself more liable than a healthy one to become the seat of tubercular disease. And not only is tubercle locally infectious, it is constitutionally so as well, and thus in any given case there is a possibility of general dissemination, with production of tubercles in all parts of the body. In such cases of **general tuberculosis** there is commonly some pyrexia, and the termination is usually fatal.

Scrofula is a constitutional condition in which there is a tendency to local chronic inflammations whose special characters are—that they persist long after their exciting cause has passed away; that they extend to neighbouring parts; that the cell exudation constantly tends to caseate and to form a fatty pulp, or else to break down into ill-formed and curdy pus; and that healthy reparative processes are conspicuous by their absence. It would appear that the tissues of a scrofulous individual are ill-formed and of low vitality, and that any inflammation, once started in them, is liable to persist on account of the inability of the tissue to initiate an acute suppuration which might rid it of its disease, or a proper reparative process which might repair the original injury. In all scrofulous inflammations the adenoid or lymphatic tissues seem to be particularly

implicated; thus, in the tonsils, pharynx, and intestines it is the lymphoid tissues which are specially diseased, and, although more difficult to demonstrate, it is yet tolerably certain that in the "strumous" or scrofulous diseases of bones and joints the same rule holds good.

An examination of a tissue in a state of scrofulous inflammation shows a considerable quantity of inflammatory new formation, with swelling and softening of the affected part; at a later stage there is much pulpy, friable matter, and finally the diseased tissue is found infiltrated with caseous material. There are typically no microscopic or naked-eye tubercles, as in tuberculosis. A microscopical examination shows the usual cell exudation met with in all inflammations, with little or no formation of fibrous tissue, and with much fatty degeneration. In addition, in many instances, primitive tubercles with giant-cells and lymphoid reticulum are found, and are quite indistinguishable from the similar growths in the macroscopic tubercles already described. The tubercle bacillus also is to be seen in some instances, but, even when it cannot be demonstrated, the tubercular nature of the lesions can be shown by inoculation of suitable animals and the development in them of tuberculosis.

The scrofulous constitution may be either congenital or acquired. Usually it is the former, but there can be little doubt that insufficient food and bad hygienic conditions may produce it in an individual in whom no congenital taint has been apparent, and there is experimental evidence to show that a predisposition may be artificially induced in animals. Thus, white mice, which are naturally refractory, lose their immunity if they be kept for some time at a temperature of 35° C. The parents of scrofulous children are often themselves scrofulous, and in other cases have been the subjects of tubercular diseases; the children of consumptive patients are frequently scrofulous. It would, however, be an error to suppose that in such cases tubercle bacilli are transmitted to the offspring, for although this is possible, it is certain that such an event is exceedingly rare,

and that which is inherited is a taint or predisposition to disease, and not the disease itself.

The connection between scrofula and tubercle is a subject which has been much discussed, and even at the present time some difference of opinion exists. There can be little doubt that the diseased conditions are closely allied, but it is practically certain that the clinical course of those cases in which grey miliary tubercles are present differs very materially from that of the others, or scrofulous ones, in which there is simply a low form of chronic inflammation without the formation of such structures. The tendency to dissemination in the former is infinitely greater than in the latter, and is a practical point of some importance.

It must, however, be allowed that in their microscopical characters many scrofulous lesions are not to be distinguished from those which are truly tubercular, and it is contended by some that the diseases are of identical nature. In support of this conclusion it is argued that tubercle has been produced in animals by inoculation with matter from a scrofulous inflammation, and that, on the other hand, scrofulous disorders have resulted from inoculation with tubercle. It has also been demonstrated that the tubercle bacillus is present in some at least of the scrofulous lesions, and those who consider that this is the cause of all true tubercle hold that its presence in scrofulous inflammations suffices to place the two processes under one classification. It is quite certain that some at least of the lesions of scrofula are of a truly tubercular nature, but it is by no means proved that all of them are; *e.g.*, the eczema of the head and face, the inflammation of the eyelids, the strumous ulcers of the cornea, and the enlargement of the tonsils and lymphatic tissue in the pharynx, have nothing tubercular about them. It must always be remembered that the general health of scrofulous children is at best feeble, that they are weakly and ill-nourished, with stomachs and intestines often out of order and unable to properly digest and assimilate the food they receive. Such patients are of course,

independently of their scrofulous taint, more liable than healthy children to such troubles as frequently recurring eczema, chronic catarrhs, and many other affections which, although common in strumous subjects, are most likely more dependent on the general ill-health of the patient than on his strumous diathesis.

It is very probable that the formation of tubercles is in many cases secondary to an inflammation which originally was chronic simply because the tissue in which it commenced was badly nourished and lowly organised. Thus, the inflammation would in such a case be primary, and the formation of tubercle secondary. This actually happens in some cases of phthisis, where the original trouble is an attack of broncho-pneumonia or pleurisy, and the tubercle is subsequently developed in the structure which has been damaged by the inflammatory process. There can, indeed, be little doubt that the spread of tubercle depends to a great extent on the condition of the tissues of each individual. All persons are not equally predisposed to it, and all tissues in the same person are not equally liable to attack. It may well be that in certain scrofulous inflammations the development of tubercle at the seat of disease is not followed by general affection of the viscera because the latter are not in a sufficiently susceptible condition, whilst in other cases, where dissemination and formation of microscopic tubercles are present, the conditions for spreading are more favourable.

Again, in those cases in which a tubercular disease of a particular organ runs its course unchecked until the death of a patient, and yet is limited throughout to that viscus—*e.g.*, the lung—in which it started, it is certain that many other viscera must have been exposed to the infection and yet have escaped, a result which must depend upon their own power of resistance. It seems, indeed, the most simple explanation of scrofulous inflammations to attribute their occurrence to the same cause as that of tubercle—*i.e.*, the tubercle bacillus, and to explain the difference in result

by the greater powers of resistance of the tissues themselves.

MANIFESTATIONS OF SCROFULA.

The scrofulous constitution is supposed to show itself in the features and build of the subject. In the so-called "sanguine" variety the skin is thin and transparent-looking, the complexion is clear, the hair is fine and light-coloured, the eyelashes are long, the eyes are large and bright, the face is oval, the bones are small, the muscular development slight, and adipose tissue scanty. In the "phlegmatic" form the skin is thick and the complexion muddy, the lips and nose are thick and prominent, the hair is coarse and dark, the eyelashes are long, the eyes dark, the features are badly modelled, and the bones and limbs are clumsy and thick. In many cases the typical appearances are by no means so strongly marked as the above summary would indicate, and between such typical cases and those in which the patient merely exhibits the usual signs of weak and delicate health all varieties may be found.

Among the many ailments to which scrofulous patients are liable the following are some of the commonest. Further descriptions of the most important ones will be found in detail in the chapters devoted to special parts of the body, such as the bones, joints, &c.

The skin is frequently the seat of chronic eczematous inflammations, which are easily excited by the slightest causes, and are most common about the exposed parts, notably the head and face.

Ulcers also are of common occurrence. They are typically of irregular shape, but with a tendency to become circular. They are variable in size, and their base is either sloughy, smooth, and shiny, or covered by pale and flabby granulations of irregular size, but usually larger than natural. The pus secreted is scanty, shreddy and watery. The edges are thin, ragged, and undermined, and both they and the surrounding skin are often blue, cold, and congested. Chil-

blains are common in scrofulous subjects, and lichen and impetigo may also occur.

The disease of the skin which is most typical of scrofula is **lupus,** which must, indeed, be regarded as essentially a scrofulous lesion. There are two varieties of lupus—**Lupus vulgaris** and **Lupus erythematosus.**

Lupus vulgaris is most commonly seen on the face, and is specially liable to attack the nose ; it usually commences in the young, and rarely begins after the age of five-and-twenty, although it may continue to *recur* at any age. Its earliest appearance is in the form of a small pimple, and in other cases there is a collection of papules or tubercles. The epidermis is thinned and shiny, and beneath it, in the cutis, can be seen a semi-translucent growth of a brownish-yellow tint, which has been compared to apple-jelly. The affected skin is redder than natural, and the epidermis soon peels off and exposes a raw red surface, which slowly ulcerates. In some cases the edge of the lupoid ulcer is surrounded by a ring of small papules like that which preceded its formation, and by a constant breaking down of these growths the ulcer increases in size. The discharge from the ulcerated surface is watery pus, mixed with epithelial scales, and readily forms a scab, around the edge of which ulceration progresses.

As in other surface lesions, the inflammatory products tend to degenerate, and the tissues around the edge of the ulcer become very soft and friable. The ulceration is slow, but may extend over wide areas and involve deep structures. Thus, the nose, eyelids, cheeks and lips may be implicated, and the nasal cartilages and bones may be destroyed. Even if the morbid process has ceased, it is peculiarly liable to recur, and treatment of all kinds not infrequently fails to arrest its progress. If the ulcers heal, the scars are usually thin and badly formed, whilst cicatrisation may progress in one part and the ulcer may extend in another ; the scars, when formed, are very liable to again break down.

Microscopic examination of a lupus patch shows chiefly

cell exudation into the papillary layers of the true skin, with desquamation of the surface epithelium and enlargement of the inter-papillary epithelial processes; giant cells are occasionally present. The sebaceous glands are larger than natural, and their cells are in a state of proliferation. The exudation around the hair-follicles generally causes the destruction of the hair. Tubercle bacilli are occasionally present, but are in any case extremely few and difficult to find.

Lupus erythematosus seldom occurs in children, and chiefly attacks females of middle age. It commences as an erythematous eruption over the bridge of the nose, and extending symmetrically to the cheeks thus acquires a shape which has been compared to that of a butterfly. The affected skin becomes thickened and infiltrated with inflammatory products, the epidermis is shed in branny scales, and finally scar tissue is formed in the substance of the derma and causes contraction of the whole area of affected skin.

The mucous membranes in scrofulous subjects are peculiarly liable to catarrh. Thus, such patients are more than others predisposed to "catch cold." The catarrh of the alimentary tract is made evident by the foul breath, furred tongue, and weak digestion. Stomatitis and discharges from the nose are of frequent occurrence, and enlargement of the tonsils, with or without catarrh of the middle ear, is one of the most common of all the lesions met with in struma. Phlyctenular ophthalmia and tinea tarsi occur in connection with the conjunctiva; from the vagina muco-purulent discharges are common, and the bladder may be irritable and unable to retain urine for any length of time.

The bones and joints are as liable as the soft parts to scrofulous inflammations, but their diseases, as well as those of the **genito-urinary organs,** will be described more at length in the chapters on such subjects.

The lymphatic glands are specially liable to scrofulous inflammations, and it has been pointed out by Mr. Treves

that the most frequent cause of their enlargement is "a lesion that implicates the adenoid tissues of a mucous membrane." The mucous membranes of the naso-pharynx and of the intestinal and bronchial tracts are specially rich in adenoid tissue, and it has already been mentioned that catarrh of these surfaces is very common. So, therefore, is disease of those glands common into which the lymphatics of these parts empty. The glands of the neck, thorax, and mesentery are infinitely more often diseased than are those of all other parts of the body, and it is quite unusual for glandular affection to follow strumous ulceration of the skin of the extremities or caries of bone.

The diseased glands become enlarged very slowly as a rule, but occasionally their growth is comparatively rapid; they vary in size, and sometimes altogether subside; after an indefinite length of time they become adherent to the surrounding parts, and evidences of inflammation slowly develop. The skin becomes dull red and adherent, suppuration ensues, and the resulting abscess opens through an irregular ragged aperture, which is very slow to heal and leaves an ugly scar; suppuration does not occur in more than a small percentage of all cases. The pus in such abscesses is ill-formed, curdy, and shreddy, and is mingled with caseous material. Very often these glandular swellings cease to grow, and remain stationary for many years; in such instances calcification may ensue, and the whole gland may be converted into a chalky mass.

On section, a strumous gland is softer than natural, and generally presents several masses of caseous material embedded in its substance; glands which have been long diseased may be completely converted into a caseous pulp, with which is mingled, in some cases, a certain amount of ill-formed pus.

Microscopical examination shows tubercle in addition to the changes common to all chronic inflammations. The cells in the gland multiply at certain places which appear to be foci of the scrofulous process, and at the same time that

they multiply they often become larger and more translucent than natural; mingled with the cells is some fibrinous exudation. In the centre of each of the foci above mentioned true elementary tubercles with giant-cells and a lymphoid reticulum appear, fatty degeneration of the cellular elements soon ensues, and caseous masses are formed. In some cases, where the influence of the scrofulous diathesis is less evident, there is a formation of fibrous tissue, though this is seldom a prominent feature.

Verruca Necrogenica.—This name has been applied to a warty growth which especially affects those who have post-mortem work to do, and is more often named "post-mortem wart." It is usually seen on the knuckles, and originates either in an abrasion or in a pustule. When fully developed it appears as a raised, moist, warty mass, the papillæ of the derma being much enlarged and often exquisitely tender. There is in some cases a covering of dry, cracked and blackened epidermis, but this is from time to time separated by a drop of semi-purulent secretion beneath it, and the raw, tender surface of the papillary layer is thus exposed. The warty condition tends to spread slowly, and may persist for months or years. It is quite certain that some of these "post-mortem warts" are really tubercular nodules, for bacilli have been cultivated from them, and tubercle of the neighbouring lymphatic glands has developed in a few cases. It does not, however, follow that all such growths are due to tubercle.

CHAPTER XV.

SYPHILIS.

VENEREAL SORES.

Venereal sores are of two kinds—(*a*) the non-infecting; (*b*) the infecting.

The **non-infecting sore** makes its appearance from two to five days after exposure to contagion, and frequently several sores result from a single inoculation.

It commences as a pustule, which bursts and discharges its contents, leaving an ulcer. The latter is generally circular in shape, sometimes no larger than a split pea, but frequently extending over a considerable area of skin; the base is grey and sloughy, with marked absence of granulations; the edges are sharply cut—giving a punched-out appearance to the sore—but are sometimes ragged and undermined; the secretion is scanty, and consists of shreddy and watery pus. Ulcers such as these may occur on any part of the penis, but are most common on the glans and on the foreskin immediately behind the corona. In many cases the lymphatic glands in the groin inflame and suppurate, the pus which is formed in them being capable of producing by inoculation sores similar to the original ulcer. As a rule, non-infecting sores are not indurated, and are therefore spoken of as "soft sores"; sometimes, however, they do become indurated, especially when they extend into the areolar tissue beneath the skin. Venereal sores of all kinds are more liable than are most ulcers to take on a phagedænic action, and by this process considerable portions of the penis

may be destroyed. Soft sores do not cause any constitutional infection, and are therefore called non-infecting.

Infecting sores are the result of inoculation with syphilitic virus. An infecting sore never makes its appearance earlier than ten days after exposure to contagion, and most commonly does not show itself for about three weeks, whilst as long an interval as six or seven weeks may elapse between infection and development of the sore. The infecting sore presents three chief varieties:

(1) The epidermis may appear abraded or excoriated, and, peeling off in flakes, exposes a circumscribed patch of a livid purple colour. There is no ulceration and no induration.

(2) An indurated tubercle may form beneath the epidermis, and, the latter remaining intact, the surface of the sore is not excoriated and does not discharge.

(3) A definite raised nodule may develop in the substance of the skin. It is coin-shaped, being flattened on the surface, with circular margins, and a definite elevated edge; it is very hard, and feels like a piece of cartilage set in the skin. The surface is red, raw, and excoriated; it discharges a thin watery fluid which contains much epithelial débris and a few leucocytes. An infecting sore such as this is a so-called "Hunterian chancre," or "hard sore."

Of these three forms of infecting sore, the second and the third are the most common, and the latter more so than the former. The most usual situation for all of them is the prepuce just behind the corona glandis. They are commonly single, but may be multiple. Any of these sores, if irritated by dirt, by friction of the clothes, or by other agencies, may suppurate, but none of them normally suppurate or form ulcers when first developed, although at the time when constitutional symptoms begin, it is by no means uncommon for the primary sore to inflame or to secrete pus. The induration is the result of inflammatory exudation into the tissues, and, in cases which are not treated, may persist as long as six or nine months, or even longer. In cases of infecting sores

the lymphatic glands in the groin become enlarged and indurated. Often they are neither inflamed nor painful, but if the sore itself suppurates from exposure to irritation of any sort then the glands in the groin may also inflame and suppurate; if pus is formed in them, it has no specific properties, and is not inoculable.

It must constantly be borne in mind that a patient may have exposed himself to the contagion of both infecting and non-infecting sores, and in not a few cases both varieties of sores develop in the same patient.

The non-infecting and the infecting sores are now generally held to be the result of inoculation with different poisons. Thus, a soft sore will by inoculation always produce a soft sore, and an infecting sore will always produce a sore which is followed by constitutional symptoms. A non-infecting sore cannot produce an infecting one.

SECONDARY SYPHILIS.

Syphilis is a specific and contagious disease propagated by local contamination.

An infecting sore, such as already described, is the usual precursor of constitutional infection, but it must, however, be clearly understood that by the time this primary sore appears the system is already contaminated, and that excision of the chancre is therefore useless as a preventive measure. The sore itself may justly be considered as the first expression of the constitutional disease.

Further local evidences of constitutional infection generally appear at about the sixth week after the development of the primary sore, and are sometimes accompanied by slight continued pyrexia. These early manifestations of syphilis are liable to succeed one another for a space of about a year or eighteen months in untreated cases, and to this stage of the disease the name of secondary syphilis has been applied. In typical cases, after apparent complete subsidence, further local lesions make their appearance, and to this stage the name of tertiary syphilis is given.

It would, however, be a grave error to suppose that these two periods and their accompanying lesions are always clearly separated from one another. This is by no means the case, for lesions usually described as tertiary may supervene within the first twelve months after infection, and secondary symptoms may occur after several years. It is practically certain that the varying severity of the manifestations of constitutional syphilis is not mainly dependent upon any alteration in the character or dose of the original virus, but rather upon the constitution of the patient in whom the disease occurs. Thus, I have seen extensive rupial ulceration with destruction of parts of the lips and cheeks occur in a woman within six months of infection by her husband, whilst the latter had himself only suffered from slight sore throat and a transient skin eruption. Again, there are some men who, although frequently exposed to contagion, never acquire the disease, whilst others are not protected, as are most, by one infection, but contract the disease afresh at some future time. In this respect syphilis merely resembles many other diseases, for there are plenty of cases in which persons are so susceptible to the infection of measles and scarlatina that they contract them as many as three or four times.

But although constitutional syphilis differs much in severity in different cases, there are certain local lesions which typically occur in the early or secondary stage of the disease, and although at first sight they appear to differ very widely from one another in their nature, such is not really the case, for in almost all of them the same pathological process is to be found. The typical lesion of secondary syphilis is **plastic inflammation.** The induration of the infecting sore is due to the exudation of plastic lymph; and whether it be the testis or the eyeball, the arterial system or the skin, the periosteum or the lymphatic glands, which is the seat of early syphilitic disease, in each and all alike there is an exudation of inflammatory products, a tendency to the formation of young fibrous tissue,

and a consequent thickening of the diseased part. In addition to these plastic exudations, ulceration occurs with considerable frequency in some tissues, but it should again be remembered that the constitution of the patient himself has much to do with the determination of suppuration.

It is not possible to deal at all exhaustively with the lesions of constitutional syphilis, and it is not proposed to do more than describe very briefly those which are the most important.

The cutaneous system suffers very constantly in syphilis, and is often the seat of the most early evidences of constitutional infection. The eruptions that occur are various. One of the commonest is a roseola, which commences on the abdomen and thorax, extends afterwards to other parts of the body, and usually fades without treatment in the course of a few weeks. In such an eruption there are numerous rose-coloured, circular spots, which are sometimes so closely set that there appears to be but little unaltered skin, while in other cases they are very few and faint in colour. The spots are not at all raised, and readily disappear on pressure.

Mingled with this roseola there is frequently seen a papular eruption, consisting of numerous small papillæ of a copper colour, which terminate either by desquamation or resolution.

At a later period tubercles may be developed. They differ from the papules chiefly in their size, and, like the latter, are the result of plastic exudation into the papillary layer of the skin. The epithelium covering these tubercles almost always desquamates, and comes away in very fine, powdery scales. After desquamation, the tubercular eruption assumes a dull-red or copper colour. In some patients the tubercles ulcerate, in others they do not, but in either case permanent pigmented scars are frequently left. The forehead—and face in general—is one of the favourite sites of these tubercles, the resulting pigmented scars being very unsightly.

Another rash, which is one of the earliest to appear as well as one of the latest to persist, is the squamous syphilide, to which the name of syphilitic psoriasis is often applied. The most common sites of the eruption are again the abdomen and thorax, and the flexor aspects of the arms and forearms. The rash consists of circular spots, slightly raised from the surface, sometimes white from the presence of epithelial débris, and, when the latter has been cast off, of a dark-reddish tint. The palms of the hands and the soles of the feet are affected with comparative frequency, and in these situations the eruption is most persistent and intractable. The squamous syphilide can generally be readily diagnosed from simple psoriasis, for there is often some other evidence of syphilis; it does not occur, as the simple form does frequently, in children; the elbows and knees, common sites for psoriasis, are usually not implicated; the rash is common on the flexor surfaces and on the palms and soles; it is sometimes mixed with other of the syphilitic eruptions already described, and in its later stages assumes the typical coppery colour.

Pustular eruptions are not common in secondary syphilis, but one form is met with sufficiently often to deserve mention. This is the syphilitic ecthyma, in which pustules form in the skin and come to the surface and discharge their contents without there being at any time marked pain or surrounding inflammation. After the pustule has burst, a scab forms of a circular shape and considerable thickness; it is very adherent, and often persists for a long time. If removed, a circular sloughy ulcer is exposed, which, when not treated, again becomes scabbed over, and shows but little tendency to heal. When cicatrisation does occur, a circular and permanent scar remains.

Such is a very brief description of the most common forms of skin eruption met with in secondary syphilis, but, although they have been treated of separately, it should be borne in mind that one of the chief characteristics of syphilitic rashes is that they differ in different parts of

the body, and in any individual case of syphilis several of the above varieties may be present at one and the same time.

Most of the eruptions are liable to assume the copper tint after lasting for some weeks, and scars, if formed, are specially liable to be pigmented.

The cutaneous appendages—the hair and nails—suffer together with the skin itself. The hairs become loosened in their sheaths by an extension to them of some of the inflammatory processes in progress in the skin, and are shed in great numbers. In some cases the head becomes completely bald, and the hair of the face may share in the general destruction. In most cases, baldness is not permanent, and after a time the hair becomes as thick as before.

The nails are liable to be attacked by a low form of inflammation which extends to the matrix and causes much swelling and tenderness of the whole finger-end. The ensuing ulceration is most chronic and intractable, and generally continues until the nail has been removed by the surgeon, for although it is loosened by the ulceration, it is comparatively seldom entirely separated.

The mucous surfaces.—At the same time that the eruptions appear on the skin, the mucous membranes are commonly the seat of syphilitic inflammations.

On **the anus**, at the junction of the mucous lining with the cutaneous surface, "mucous tubercles" develop. These are warty growths with broad bases of attachment, covered by a thin layer of epithelium, and moist from the constant watery discharge which exudes from them. They are commonly multiple, and, when excoriated or inflamed, give rise to considerable pain. They consist of young connective-tissue, and vary in size from that of an ordinary cutaneous wart to that of a hazel-nut.

The lips are also liable to be attacked by similar growths, which seldom attain so great a size as do those around the anus. In addition, the lips are often cracked,

fissured, and superficially ulcerated. **The gums** may be in a similar state.

The tonsils are ulcerated in almost every case of acquired syphilis. They become swollen and inflamed, and ulcers of circular shape with sharply cut edges penetrate deeply into their substance; the base of these ulcers is often sloughy. Mucous tubercles also are common on the tonsils.

The tongue is sometimes involved in the eruptions which occur on the cutaneous surfaces, and is specially liable to be attacked by the squamous syphilide; mucous tubercles, and superficial ulceration are common around its edges, but deep ulceration is rare.

The larynx may be simply inflamed, and its mucous membrane slighty swollen, or mucous tubercles and superficial ulceration, such as occur on the lips and tongue, may be present, though they are very rare. It is to the diseased state of the larynx rather than to that of the tonsils, that the hoarseness which so often occurs in secondary syphilis is to be attributed.

The nose is not often affected in early syphilis, but its mucous lining is occasionally inflamed at the same time that the lips and gums are attacked.

The eye.—The commonest affection of the eye in secondary syphilis is plastic iritis. This is accompanied by pain and aching, with more or less photophobia and lachrymation. An examination shows that the conjunctiva is hyper-vascular, and the iris is surrounded by the deep zone of vessels which is the usual accompaniment of all forms of iritis. The aqueous humour is turbid from admixture with inflammatory products, and the colour of the iris is consequently blurred and altered in shade. The pupil is sluggish, often different in size from its fellow, and sometimes irregular. In the anterior chamber, on the margins of the pupil, and in the substance of the iris may be seen beads of lymph. If syphilitic iritis be allowed to run its course unchecked, the plastic effusion is liable to

become organised, so that the pupil is either blocked by fibrous tissue, or, more frequently, the iris becomes adherent to the lens capsule on the one hand, or to the cornea on the other. The formation of such adhesions is often followed by a very persistent form of chronic iritis, which sometimes results in a gradual extension of the inflammation to the ciliary processes and choroid.

The testes are often attacked by plastic inflammation; usually in the second year after the commencement of the disease; one or both glands may be involved. In this form of orchitis there is a general enlargement of the testis from effusion into it of plastic exudation, which tends to become organised into fibrous tissue, but which is generally in great part finally absorbed even in cases which are not subjected to treatment; on section, such a testis will appear more tough and fibrous than a normal organ. In these cases the inflammation commonly extends to the tunica vaginalis, and effusion of fluid into the cavity of the latter is of frequent occurrence. The epididymis is not usually implicated in the inflammatory process, and, except for slight œdema, the cord is also free. Such a testis is painless, and gives no trouble, except by its size. It is often as large as a hen's egg, smooth, heavy, oval, not tender, with marked absence of testicular sensation when subjected to pressure, and firm to the touch. In cases of long duration, the gland substance may be compressed by the contraction of the fibrous tissue, and become atrophied. The organ thus passes into a condition of cirrhosis entirely analogous to that which is met with in the liver of drunkards.

The diseases of the **bones**, **joints**, and **arteries** are described in the chapters devoted to these subjects.

TERTIARY SYPHILIS.

The typical lesion of tertiary syphilis is the **gumma**, but, in addition, there is a general tendency to suppurations in various parts of the body.

A **gumma** is an inflammatory new growth—an infective granuloma—in which the cell exudation has a special tendency to degenerate and form fatty or caseous masses. The exudation at first is in no way distinguishable from that of a simple inflammation, and its initial tendency is to develop into young fibrous tissue. This development, however, is generally imperfect, for the cell-growth is not sufficiently vascularised, and soon degenerates into a caseous pulp. The caseous material is always found in the central or oldest part of the gumma, and is surrounded by the ill-developed fibrous tissue just mentioned, whilst outside this, again, the cell proliferation progresses, and causes a constant increase in the size of the swelling; scattered through the cell infiltration "giant cells" are often seen, they are, however, not nearly so numerous as in tubercle, and usually have neither so many nuclei or cell-processes as the tubercular giant cells. Three zones are commonly described in a gumma—the outer or cellular, the middle or fibrous, and the central or caseous.

To the naked eye a gumma presents on section the following appearances:—It is a growth of a white or yellowish-white colour, and tends to be globular in form. Although it may be sharply circumscribed, it is nevertheless not encapsuled, and in some cases it infiltrates the surrounding tissues; its consistence varies; in some cases a gumma is tough and leathery, in others soft and pulpy and mingled with the products of suppuration.

As a gumma increases in size it is accompanied by all the symptoms of chronic inflammation, and is often surrounded by much plastic exudation. When it comes to the surface and discharges its contents, a circular crater-like cavity is left, the edges of which are much undermined and the base sloughy. If no treatment be adopted, healing is very slow, and a depressed scar always results. Gummata vary much in size, the average growth in the tissues of the

being not larger than a walnut, while swellings as large as or larger than an apple are sometimes seen. The commonest sites for gummata are the muscles and subcutaneous tissues, but they occur also in the liver, kidneys, spleen, brain, spinal cord, tongue, bursæ, joints, and testes. In addition to the definite circular swellings to which the name of gumma should be limited, inflammations with a tendency to the same formation of caseous matter, but without the same circumscription or tendency to form globular masses, are common in tertiary syphilis, especially in connection with the osseous system. To such inflammations the epithet "gummatous" is well applied.

Diseases of the skin and cellular tissue.—Ulcers of a peculiarly unhealthy and persistent nature are of frequent occurrence in tertiary syphilis. They may form in any part of the body, but are perhaps most common on the lower extremities. The base of such an ulcer is usually sloughy, and the pus secreted scanty and shreddy. The edges are sharply cut, and are often crescentic in shape; these ulcers sometimes attain a considerable size, and when occurring on the face may simulate lupus.

Rupia is a form of the ulceration of skin which is met with in syphilis alone. Each ulcer commences as vesicle; this pustulates, its contents are discharged, and a scab forms. Beneath the scab ulceration progresses eccentrically, and fresh scabs are produced. In consequence of the constant increase in circumference of the ulcer, the more recent scabs are larger than the earlier ones, and a conical concretion is thus formed, which has been aptly compared to a limpet-shell, the first and smallest scab being at the apex of the cone. If the scab is removed, an unhealthy, sloughy ulcer of circular shape, and about the size of a sixpence or a shilling, is exposed. When this has healed, a pigmented cicatrix remains. A score or two of such ulcers may be present at the same time in a single individual.

Diseases of the mucous surfaces.—The tongue may be the seat of gummatous growths or of deep ulceration.

The former occur chiefly on the dorsum, and often near the tip. Their size varies from that of a pea to that of a walnut, and, when their contents have discharged, a typical gummatous ulcer remains. The deep ulceration may affect the whole dorsum of the tongue, or a part only of one of the lateral margins. In the former case fissures form, and the whole surface becomes cracked and furrowed. In the latter, deep ulcers, with sharply-cut edges and sloughy bases, develop on the sides of the tongue, chiefly towards its posterior part, and sometimes closely simulate epithelioma. Both these forms of ulceration are most persistent and intractable, and are, moreover, liable to frequent recurrence.

The larnyx, like the tongue, is liable to be destroyed in part by ulceration. This very frequently commences in the epiglottis, and may completely destroy it. The ary-epiglottic folds are also commonly involved, and the destructive process may extend to the true vocal cords. In bad cases the cartilages of the larnyx are attacked, and either ulcerate or necrose. In consequence of the very considerable formation of fibrous tissue which follows the ulceration, the glottis is sometimes stenosed, and dyspnœa results.

The alimentary tract, as a whole, is remarkably free from syphilitic disease, for although cases of ulceration of the small intestines have been recorded, the latter, as well as the œsophagus and stomach, usually escape. The rectum is not so fortunate, and extensive ulceration spreading up from near the anus, sometimes causes the formation of numerous fistulæ, with thickening of the gut and resulting stricture. In some cases a fatal result ensues either from long-continued suppuration or from peritonitis.

Visceral syphilis.—This subject is too large to be dealt with at any length in the present work, and it must suffice to mention briefly the chief changes that are found.

The lungs and heart are not often affected, but the former may be the seat of fibroid induration, and in the latter organ gummata may develop.

The abdominal viscera are more frequently diseased. In

the liver and the spleen gummata are common, and in the former viscus the capsule may become thickened by perihepatitis, and bands of fibrous tissue may develop in its substance; by the connection of these bands the liver becomes puckered, and its surface deeply fissured and irregularly lobed. Gummata may form in the kidneys also,

FIG. 18.

Section of a Syphilitic Testis, with gummata of various sizes.

but are less common than in the liver and spleen. In all these viscera there is often, in addition to other lesions, amyloid degeneration.

Both the brain and spinal cord are liable to gummatous disease, and sclerosis may result from the formation and contraction of fibrous tissue. The peripheral nerves also are occasionally the seat of syphilitic inflammation.

In the testes gummata are met with; they occupy the body of the gland rather than the epididymis, and are prone to break down and discharge through the scrotum. A testis with gummatous growths is in the early stage nodular and of irregular shape, and later on is often in

great part destroyed; both testes may be affected. (See Fig. 18.)

The syphilitic diseases of the eye are both numerous and destructive; the most important are diffuse inflammation of the choroid and retina, and chronic inflammation of the iris and ciliary region.

The diseases of the bones, joints, and vessels in tertiary syphilis are described in the chapters devoted to these subjects.

CONGENITAL SYPHILIS.

Syphilis may be transmitted to the offspring by either parent, and, when inherited from the father, the mother may herself become infected from the child without the occurrence of any primary sore. It is indeed certain that in practically all cases where the child is infected by the father's semen the mother is also infected through the child, and it has been observed that a syphilitic infant with sores about its mouth never infects the nipples of its mother, although it may inoculate the nipples of another woman, thus proving that the mother is insusceptible because already syphilitic. This is known as "Colles' law."

A syphilitic infant frequently shows no evident signs of disease at the time of birth, and none may appear for several weeks. In other cases the child is evidently syphilitic when born. The disease often results in the death of the fœtus whilst *in utero*, a fact which readily explains the frequency of miscarriage in mothers who are pregnant by syphilitic husbands.

One of the earliest symptoms of congenital syphilis is a difficulty in breathing, which has earned for the disease the popular name of "the snuffles." This is due to inflammation, and possibly ulceration, of the mucous membrane of the nose, with consequent catarrh and obstruction to the free entry of air. In bad cases, on account of the interference with the nasal respiration, the child has great difficulty in sucking.

Within a few weeks **eruptions** appear on the cutaneous and mucous surfaces, especially on the buttocks. The eruption differs in different cases, and in different parts of the body in the same case. Sometimes there are rose-coloured spots, and with these may be mingled others of a distinct coppery colour. On the palms of the hand and soles of the feet the eruption is generally squamous, and the epidermis desquamating. Around the anus, in the groins, and at the angles of the mouth, mucous tubercles are common. Occasionally the eruption is vesicular, and large bullæ are formed; to such a rash the name of "syphilitic pemphigus" is applied. The whole skin is, in bad cases, much wrinkled, dry, and dirty, and the child looks prematurely old and wizened. Cracks and fissures are common about the angles of the mouth, and often leave pale radiating scars, which permanently mark the patient and afford valuable evidence of the disease.

There is frequently considerable disturbance of the gastro-intestinal tract, with tumid abdomen and diarrhœa. The abdominal viscera may be enlarged either by amyloid disease or by simple congestion and fibroid thickening. The milk-teeth are late in being cut, and are liable to early decay; they do not, however, exhibit any change which is typical of syphilis.

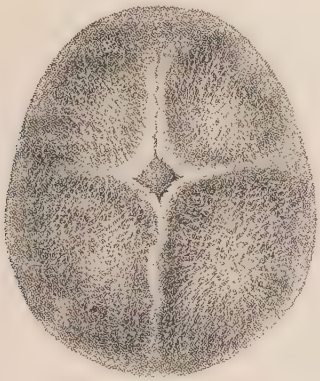
The **osseous system** exhibits lesions which are amongst those most characteristic of the disease. They have been especially described by Drs. Barlow and Lees in England, and by Professor Parrot in France. These lesions are of two kinds—(1) **atrophic**, (2) **osteophytic.**

In the bones of the skull the atrophic lesions are of two varieties. In the one the bone is not diminished in thickness, but its substance undergoes a degenerative change, with the production of a gelatinous-like material, the bone-salts being removed. In the other there is simple atrophy of the cranial bones at those parts where they are exposed to the greatest pressure, the bone being thinned away until the brain is only covered by a transparent mem-

brane. In this condition—which is almost exactly similar to that which is met with in rickets, and to which also the name of "craniotabes" is applied—the soft places in the skull can be readily felt by the finger during life; this is not the case with the gelatiniform degeneration.

The osteophytic lesions of the skull are very typical, and are the result of a chronic periostitis. In them there is a production of new bone from the periosteum, with consequent thickening of the calvaria. This thickening occurs

FIG. 19.



Skull from a case of Congenital Syphilis, showing the symmetrical bosses over the frontal and parietal bones which result from periostitis.

in very definite situations, and is almost invariably symmetrical. The sites for the thickening are the parietal and frontal bones on each side of the middle line, and in close proximity to the anterior fontanelle. In these situations, rounded, raised bosses of bone are formed, which sometimes attain a thickness of half an inch or more, and produce a most peculiar and characteristic appearance. Skulls so altered in shape are frequently called "natiform."

In the long bones the atrophic lesions are limited to the epiphysial ends. They are characterised by excessive

calcification of the cartilage matrix, and the consequent formation of a very imperfect bone, and by the absorption of the bone already produced. In consequence of these alterations in structure, the attachment of the diaphysis to the epiphysis becomes weakened, and complete separation is readily produced by the application of slight violence. When this separation occurs, suppuration occasionally ensues. In these cases of disease of the epiphysial ends there may be a spurious paralysis, which is dependent upon the pain caused by movement as well as on the inherent weakness of the bone. Gelatiniform degeneration, such as affects the skull, is also met with in the long bones.

The osteophytic lesions, which here, as in the skull, are of an inflammatory nature, are much more common than are the atrophic ones; the bones most commonly affected are the humerus, tibia, femur, and ulna, although no bone is altogether exempt. The formation of osteophytes is in some cases very extensive, and the osteophytes themselves present various appearances. In one variety, described by Parrot as the "osteoid," there is a production of new bone which is rather more spongy than normal osseous tissue, and is deposited in a series of layers placed one on the other, and each separated from the one beneath by a layer of medulla. In other cases the new formation beneath the periosteum is hardly at all calcified, and consists rather of fibrous tissue of a yellowish colour, slightly vascular, and simulating the animal matrix of bone. To this variety of osteophytic growth Parrot has applied the term "rachitic," but between it and the osteoid variety all grades may be found.

The above are the chief forms in which congenital syphilis manifests itself in the infant. Later in life other changes occur, which are too extensive to permit of lengthened description.

The cornea may be the seat of interstitial keratitis, an affection which commences in one eye and goes on to produce a more or less complete opacity of the whole

cornea, but afterwards tends to clear up, and generally leaves only a few slightly opaque patches. The other eye begins to be affected at the time when the eye first attacked is improving, and passes through similar stages of disease. The retina, the iris, and the choroid may be attacked by chronic inflammations.

Deafness is liable to supervene very suddenly in early childhood, and to result in complete loss of hearing. It is

FIG. 20.

Notched and jagged teeth, from a case of Congenital Syphilis.

apparently of nervous origin, and occurs independently of all external cause or of apparent change in the conducting media. Chronic inflammation of the tympanum, with thickening of the membrane, is also met with in congenital syphilis.

The **permanent teeth** are in some cases deformed in a very typical manner. The central incisors are small and ill-developed, are separated from one another by a larger space than is usual, and are deeply notched on their cutting edges. It is to this notching that the greatest importance is to be attached. The lateral incisors and the canines are brittle, and the enamel is broken away at their free edges so as to leave a portion of the dentine protruding, like the peg of a peg-top, from the rest of the

crown. All the teeth are liable to premature decay. (See Fig. 20.)

Ulcerations of the skin and **gummata** are of common occurrence in congenital syphilis; they do not differ from similar lesions in patients who have acquired the disease.

Chronic osteitis and periostitis are not infrequently met with in children of about six to twelve years of age. They affect chiefly the long bones, and in some cases attack several bones in the same patient, causing much deformity by the great thickening, as well as curvature and lengthening, which they produce.

The joint diseases of congenital syphilis are described at length in the chapter on "Diseases of the Joints."

Bacillus of syphilis. — Specific bacilli have been described by Lustgarten and Eve, but their observations are not convincing. Lustgarten's bacillus, or at any rate an organism which closely resembles it, was found by Alvarez and Tavel in the smegma of the prepuce and vulva also, but others have confirmed Lustgarten's investigations, and have shown the presence of the bacillus in the tissues. Eve's bacillus differs from Lustgarten's in many points, and it cannot yet be said that the parasitic nature of syphilis has been proved

CHAPTER XVI.

TUMOURS.

A TUMOUR is a mass of newly-formed tissue tending to grow and persist independently of the structures amongst which it is placed, and not serving any useful or physiological purpose. Thus, a tumour differs in all these particulars from a mere hypertrophy, which is commonly the direct or indirect result of local changes in nutrition, or is a consequence of increased physiological requirements, and which, moreover, instead of persisting, as a tumour usually does, is liable to subside when the stimulus which induced it has passed away. In another respect also a tumour differs from a mere hypertrophy, for, whilst in the latter the natural shape of the organ or tissue is preserved, a tumour forms a definite swelling or outgrowth, which usually spoils the normal symmetry of the part.

From inflammatory swellings a tumour differs in its tendency to persist, and not to terminate in resolution or suppuration; in its origin, which is frequently independent of all exciting causes; and in its intimate structure, which is usually materially different from that seen in inflammatory growths.

Again, a tumour being defined as a "mass of newly-formed tissue," it is evident that swellings caused by extravasation of blood, by displacement of an organ, or by distension with fluid of a pre-existing cavity, such as the tunica vaginalis or a bursa, cannot be included under this head.

CAUSES OF TUMOURS.

The causes of tumours are but little known, and even in those cases where their origin is distinctly traceable to injury or irritation we are at present quite unable to explain why in the vast majority of cases these usually produce inflammatory changes, whilst in the few they cause tumour-growth. The following theories have been advanced, and, as they all have something to support them, they may be briefly considered.

First, **inclusion theory, or embryonic hypothesis of Cohnheim.**—According to this, tumours are developed from embryonic cells, which having been produced in greater numbers than are required for the formation of the fœtal tissues, remain in the locality in which they originally grew, and are lighted up into active growth either by the application of some artificial irritation or by the normal physiological activity of the part excited in a natural manner.

In support of this theory it is pointed out that moles, which are composed of connective-tissue cells, may develop in later life—and especially if irritated—into melanotic sarcomata; that the exostoses which are common at the articular ends of long bones undoubtedly develop from portions of epiphysial cartilage which have been left uncalcified in the shaft; and that many tumours originate in parts of the body in which structures present in fœtal life have been suppressed, or in parts where, in the process of closure of clefts or involutions of epithelium, it appears probable that more cells than could be utilised might have been included. For example, epithelioma is most common at the junction of mucous membrane with skin, and in parts, like the rectum and uterus, where a junction has been effected during fœtal life between the viscera and the surface of the body. Although, however, this theory is an attractive one, it by no means accounts for even a considerable proportion of all tumours, and is not sufficiently supported by either

physiological or histological research to allow of its general acceptance.

Second, **hereditary influence.**—In a certain number of cases of tumour a clear family history of similar growths may be obtained. This of course does not prove that tumours are of constitutional origin, for they may be inherited as local affections, just as may club-foot or other deformities.

Third, **irritation and injury.**—Whilst fœtal inclusion and hereditary taint may act as predisposing causes, irritation and injury may be considered as exciting causes of tumour growth. Thus, irritation of the skin by acrid matters may produce warts—*e.g.*, in workers in tar or petroleum—or may promote epithelioma, as in sweeps' cancer of the scrotum. Again, the mechanical irritation of a mucous surface, such as that of the lip or tongue by a sharp tooth, a jagged pipe, excessive smoking, &c., may result in the production of a similar growth.

But, whilst long-continued irritation often precedes warty or epitheliomatous tumours, the occurrence of sarcomata is influenced rather by direct and sudden injury, especially in the form of contusion. Thus, a blow on the testis may precede sarcoma of the organ, and a similar history is not uncommon in periosteal sarcomata of bone.

Fourth, **physiological activity and decline.**—Sarcomata are especially prone to develop in early life in rapidly growing connective tissues, whilst carcinomata grow in glands and other structures undergoing retrograde change, such as the mamma and uterus after the child-bearing period.

Parasites in Tumours.—During the past few years much attention has been paid to the examination of tumours, and especially those which are malignant, in order to demonstrate the presence of parasites. Some observers are of the opinion that protozoa can be demonstrated in the cells of carcinoma and sarcoma; but most pathologists do not consider that the bodies in question are parasitic, and attribute

the appearances to cell inclusions, or degenerations of cell protoplasm. It must further be noted that all attempts to cultivate the supposed protozoa have failed, and it is quite certain that at present there is no proof that tumours are caused by parasitic infection.

DEVELOPMENT AND DEGENERATION OF TUMOURS.

Whatever may be the cause or causes of a tumour, when once it has developed it almost always persists, and commonly continues to grow. It is true that some growths occasionally disappear spontaneously, *e.g.*, warts and lipomata; and that others—*e.g.*, the cancellous exostoses of the long bones—after a time cease to increase in size. These, however, are exceptions; and a tumour, as above stated, is characterised both by its persistency and its tendency to increase.

In some cases the structure of a tumour undergoes changes in its further development, and, putting aside for the present the ulceration which is common in malignant growths, it may be said that these changes are generally of a degenerative nature.

Calcareous degeneration is one of the commonest changes, and is often seen in enchondromata, and more rarely in fatty tumours whose rate of increase is very slow, or which have ceased to grow.

Mucoid degeneration is also seen in cartilaginous tumours as well as in many of the sarcomata, and is characterised by a gelatinous, semi-translucent appearance, or else by the formation of a cyst containing viscid fluid resulting from liquefaction of the matrix.

Fatty degeneration is met with especially in the epithelial cells of cancerous growths, but occasionally in the sarcomata and in innocent tumours.

Inflammation is a rare complication of malignant tumours, but is still more rarely met with in innocent growths. It is characterised by the usual local signs, and

may terminate in suppuration. I have seen this in both carcinomata and sarcomata—in several cases of the former in the female breast, and in the latter in the neck and testis. It is a complication to be remembered, for the presence of pus is usually to be looked upon as contra-indicating a new growth.

Sloughing is a very rare occurrence in tumours, and is limited to a few instances of large fatty or soft fibrous masses, and to cancers.

Hæmorrhagic infiltration is often seen in soft sarcomata, and more rarely in other tumours; it sometimes causes a rapid increase in size of cystic growths, and may thus simulate malignancy.

DIFFERENCES BETWEEN INNOCENT AND MALIGNANT TUMOURS.

All tumours are, for clinical purposes, roughly divided into two large groups, the **innocent** and the **malignant**. To the special characteristics of each of these groups it will now be necessary to turn before passing on to consider the structure of the different forms of new growth.

Malignant tumours differ from innocent ones in the following particulars:—1. Mode of growth; 2. glandular affection; 3. dissemination; 4. affection of the general health; 5. local recurrence after removal.

1. **Mode of growth.**—All malignant growths tend to *infiltrate and replace* the tissues in which they develop, and in this respect differ essentially from innocent tumours, such as lipomata, fibromata, &c., which simply push the structures to one side, or *displace* them, and separate them from one another. Malignant growths are practically never thoroughly encapsuled, but blend with and are inseparable from the structures around them. Into these they grow and on these they feed, so that in time the tissues are replaced by the growth. But, in addition to infiltration, a malignant tumour *increases with much greater rapidity* than does an innocent one. Its blood-supply is large, its cell multiplication rapid, and

within three or four months it may attain a considerable size. The skin covering it is at first tightly stretched, but soon becomes red and shiny; then dimpled, puckered, and adherent; and finally, giving way, allows the protrusion of a bleeding, soft, pulpy mass. Exposure to the air is soon followed by decomposition, with foul smell and fetid secretion, and the patient is worn out by the combination of septic conditions and profuse discharge of blood and pus.

2. **Glandular affection.**— One of the best marked and most widely recognised signs of malignancy is the occurrence of secondary growths in the neighbouring lymphatic glands. Enlargement of glands near a tumour may be due to irritation or inflammation, but these have nothing to do with the glandular affection under consideration, which consists in a reproduction in the gland of the exact counterpart of the primary growth, so perfect that any slight peculiarities in the original tumour can be easily recognised in the glandular enlargement.

But although glandular affection is good evidence of malignancy, whether the glands shall or shall not be occupied by secondary growths in any given case depends on two conditions—(*a*) the character, and (*b*) the locality of the growth.

As regards the character, it may be said that, speaking generally, the carcinomata more often cause glandular affection than do the sarcomata, and that the epitheliomata are of all tumours those most likely to give rise to this complication. The sarcomata do, however, in many cases affect the glands; one variety—the melanotic—almost invariably doing so.

As to locality, carcinomata in certain situations, such as the œsophagus, stomach, and superior maxillary bone, comparatively seldom cause secondary growths in the lymphatics; whilst, on the other hand, sarcomata of the testis almost always affect the glands, though similar growths in the breast hardly ever do so. For further

information on these points reference must be made to the chapters on the diseases of the various tissues and organs.

3. **Dissemination.** –The reproduction of a growth in other parts of the body, often far removed from the seat of the primary tumour, is justly regarded as evidence of the worst kind of malignancy. No viscera are exempt, and the bones, muscles, and central nervous system may any or all be implicated. As in the case of the glands, the secondary tumours exactly resemble the primary growth, and the occurrence or absence of dissemination is dependent on the structure and position of the primary tumour. The growths which disseminate most rapidly are the round-celled sarcomata and the encephaloid cancers.

4. **Affection of the general health.**—The term "cancerous cachexia" has for long been employed to indicate the wasted form and enfeebled health which so often accompany the development of a malignant tumour; and, although the old idea that the cachexia was of a special kind peculiar to cancers is no longer upheld, there is no doubt that the general health does become affected, and that a patient often wastes to an extent which is out of proportion to the apparent demands made by the disease upon his vital resources. Emaciation becomes more marked when the viscera are implicated, and special symptoms are developed according to the particular viscus affected.

5. **Local recurrence**.—Almost all tumours which show their malignancy in one of the ways already mentioned are also prone to recur locally when removed; but many growths which do not tend to become disseminated, do tend to recur locally and sometimes to affect the glands. Such growths as these are said to have a "limited malignancy," the chief of them being the fibro-sarcomas or recurrent fibroids of the skin and subcutaneous tissues, the rodent ulcers, and to a less extent the cutaneous epitheliomata of the body and of the lips. In some cases the tumour recurs time after time in the scar left by the operation, and may, by its local extension and implication of vital structures, destroy life

without causing any glandular affection or becoming disseminated in the viscera.

CLASSIFICATION OF TUMOURS.

All tumours may be placed in one of three classes: I. connective-tissue growths; II. epithelial growths; III. cysts.

Both the first and second classes may be again subdivided into innocent and malignant, and the whole tabulated according to their structure as follows:—

I. Connective-tissue tumours—
- Innocent:
  - Fibroma.
  - Myxoma.
  - Lipoma.
  - Chondroma.
  - Osteoma.
  - Lymphoma.
  - Lymphagioma.
  - Myoma.
  - Neuroma.
  - Angeioma.
- Malignant:
  - Sarcoma.

II. Epithelial tumours—
- Innocent:
  - Adenoma.
  - Papilloma.
- Malignant:
  - Carcinoma.

III. Cysts.

In the following pages the general structure of the various growths will be described; but, as their clinical course and behaviour vary much with their position, I have chosen to deal with them in detail in the chapters on the different organs and tissues of the body.

The classification of tumours, according to their origin from epiblast, mesoblast, and hypoblast, is unpractical, and has been to a great extent abandoned.

CHAPTER XVII.

INNOCENT CONNECTIVE-TISSUE TUMOURS.

All the connective-tissue tumours are developed from connective-tissue cells, which, in the case of the innocent growths, are further developed into some structure which is an exact imitation of one or other of the various connective tissues found in the normal adult body. In the malignant growths—the sarcomata—the cells remain in a more or less undeveloped state, and form large masses such as are not met with in any part of the normal tissues.

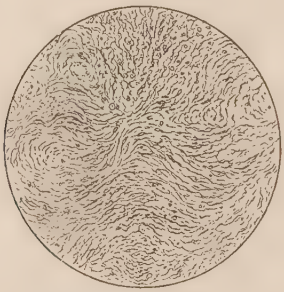
FIBROMATA.

The fibromata are tumours composed of fibrous tissue, and just as in the human body fibrous tissue may be loose and succulent, *e.g.*, in the scrotum and eyelids, or dense and firm, as in the ligaments and tendons, so also the fibromata may be soft or hard. In the one case they are composed of loose fibrillar tissue with large spaces and many cells; in the other of densely packed bundles of fibres closely interlaced. The fibromata grow slowly, and are but slightly vascular.

The soft fibromata are most common on the inner side of the upper arm and thigh, close to the axilla or the perineum, and are more rarely met with in the labia and scrotum, on the scalp, and in the subcutaneous tissue in other parts of the body. They often attain a considerable size, and may weigh several pounds. They are commonly pedunculated and pendulous. Occasionally, fluid accumulates in the intercellular spaces, and forms cysts.

The hard fibromata occur in the subcutaneous tissue on the periosteum of the jaw-bones as epulides, or on that of the bones of the naso-pharynx as fibrous polypi. More rarely they are seen in the intermuscular septa, on the nerves

FIG. 21.



Hard Fibroma from the Subcutaneous Tissue.
(Crouch : $\frac{1}{2}$ inch.)

as "false neuromata," as well as in the breast, where, mixed with gland tissue, they are called adeno-fibromata.

MYXOMATA.

Myxomata are tumours composed of mucous tissue. There are all grades between a soft fibroma and a myxoma, and the latter may be justly looked upon as a soft fibroma, the intercellular substance of which has been replaced by mucin. Such a metamorphosis, if complete, would leave only the branched connective-tissue cells, enclosing spaces containing viscid fluid, but it is very rarely that a pure myxoma of such a structure is found, all those that I have myself examined containing some intercellular fibrous tissue.

The myxomata form soft, gelatinous, semi-translucent tumours, generally pedunculated, and yielding a viscid fluid on section. The microscope shows swollen fibrous tissue, and a network formed by the branching processes of connective-tissue cells.

Secondary changes are common, for, on account of the delicacy of their structure, extravasations of blood into the substance of the myxomata are of frequent occurrence, and the thin pedicle being liable to become twisted, the tumours may inflame or slough.

Clinical characters.—Myxomata may develop at any time of life, and are almost limited to mucous surfaces, such

FIG. 22.

Myxoma from the Middle Turbinate Bone.
(Crouch : $\frac{1}{8}$ inch.)

as the nose, bladder, uterus, &c., where they form the well-known mucous polypi.

LIPOMATA.

Lipomata are tumours composed of fat. They form lobulated growths, and may attain a great size, sometimes weighing many pounds. They are surrounded by a firm fibrous capsule, which is closely adherent to the neighbouring tissues, though loosely attached to the tumour. Of this fact advantage is taken in operations for the removal of such growths, the capsule being freely opened, and the tumour easily shelled out from within it. A lipoma, on section, is of a yellowish colour, and is indistinguishable from subcutaneous fat except by the greater firmness of its fibrous matrix. Microscopical examination shows that the fat is contained in connective-tissue cells which are held

together by a delicate network of fibres. The cells are larger than those usually seen in normal fat.

Secondary changes are not common in the lipomata, but in those which are of very long standing, and especially in those which have ceased to grow, calcification is of occasional occurrence. When they are very large, and subjected to pressure, the skin over them is sometimes ulcerated.

Clinical characters.—Lipomata are generally met with in people over middle age, and in those who are stout rather than in thin subjects. They may occur in the subcutaneous tissue of any part of the body, but show a distinct preference for the dorsal surfaces of the trunk and limbs, and are in some cases apparently the result of friction or pressure. Common situations for them are the subcutaneous tissue of the neck, shoulders, lumbar and gluteal regions. Their capsules are attached by small fibrous processes to the skin, so that the latter becomes dimpled or puckered if attempts are made to raise it from the tumour. In most cases lipomata are superficial to the muscles, though occasionally they dip beneath and between the latter. Other situations in which deep-seated lipomata occasionally occur are the spermatic cord, the omentum, and the viscera; and a few rare cases have been recorded in which lipomata have occurred as **congenital** tumours attached to the periosteum of the long bones. I have seen such a growth attached to the greater part of the shaft of the femur.

Diffuse lipoma is a form of fatty tumour which is not limited by any capsule. It is simply a local overgrowth of the fat normally found in the subcutaneous connective-tissue, from which it does not differ in any respect. The patients in whom diffuse lipomata are found are usually stout men over middle age, and those who are of intemperate habits appear specially liable to be affected. The most common positions for these growths are the submental regions, where they form pendulous masses, looking like an exaggerated double chin; the back of the neck and

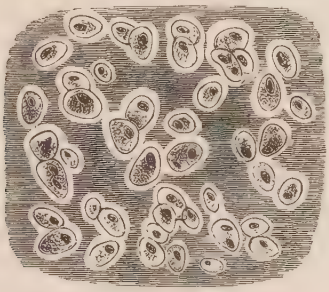
post-mastoid regions, where they are usually symmetrical; the groins, scrotum, and abdominal walls.

CHONDROMATA.

The chondromata are tumours composed of cartilage.

They vary much in size according to their position and attachments, are surrounded by a fibrous capsule, and are often nodulated. Their cut surface is divided into lobes, and is either bluish-white, smooth, and glistening, or fibrous and striated; some, again, are soft and gelatinous on

FIG. 23.



Hyaline Enchondroma from the Scapula.
(Crouch : $\frac{1}{6}$ inch.)

section, and present a granular or ground-glass appearance. Each tumour is closely attached to its capsule, which forms for it a sheath or perichondrium. Microscopical examination shows different structure in different cases. In some the matrix is hyaline, as in articular cartilage; in others it is fibrous, as in fibro-cartilage. The cells are sometimes enclosed in groups of three or four in capsules; in other tumours they are not encapsuled, and are scattered singly throughout the matrix. The cells vary much in size even in the same tumour. They are generally round or oval, but occasionally polyhedral or stellate.

Secondary changes are of very common occurrence in chondromata. The intercellular substance in these tnmours

is exceedingly prone to undergo mucoid softening, the matrix being converted into a sticky fluid, and forming cysts. Calcification is also common. It occurs especially in the more central parts of the growth, the salts being first deposited in the nuclei of the cells. Ossification is not common in pure enchondromata, but does occasionally occur.

Clinical characters.—The enchondromata are most often met with in the bones of the hand, and more rarely in the foot, the long bones, and the pelvis. They grow also from the rib-cartilages, and are specially liable to spring from two or three of the latter rather than from any single one. In the soft tissues they are found in the parotid, testis, and breast; in the latter very rarely, and in the former seldom unmixed with sarcomatous or other tissue.

OSTEOMATA.

Osteomata are tumours composed of bone. They almost always grow from some part of the osseous system, and are fully described in the chapter on "Tumours of Bone." Very rarely, osteomata are also met with in the soft tissues, and examples have been recorded of bony growths in the lungs, brain, and parotid gland. In the muscles also single bony growths are met with, whilst in the disease known as "myositis ossificans" there is a formation of bone in many parts of the muscular system.

The **lymphomata** and **lymphangiomata** are fully described in the chapter on the "Diseases in the Lymphatic System."

MYOMATA.

The myomata are tumours composed of muscular tissue, which is almost always of the unstriped or involuntary variety, and is frequently mixed with much fibrous tissue.

Myomata form rounded, encapsuled tumours whose cut surface is firm and fibrous. A myoma is softer than a hard fibroma, and generally not so white and shiny. Microscopical examination shows that the growth consists

of long spindle-shaped fibres with central nuclei, placed with their long axes parallel, and held in apposition by a framework of fibrous tissue. The myomata are but little vascular.

Secondary changes in these tumours are common, especially when they occur in the uterus; there they are very liable to become calcified.

Clinically, myomata are found in the uterus and prostate, and infinitely more rarely in the intestine, stomach, and œsophagus.

Striped muscle is met with in mixed tumours only, chiefly in sarcomata of the kidney.

FIG. 24.

Uterine Myoma.
(Crouch : $\frac{1}{6}$ inch.)

NEUROMATA.

Neuroma is a term which is usually applied indiscriminately to all tumours growing on a nerve-trunk, those which are composed of nervous tissue being called "true neuromata," those composed of fibrous, myxomatous, or sarcomatous tissue being named "false neuromata."

True neuromata are very rare. They may be composed of either medullated or non-medullated fibres, and cases have been described in which neuromata in connection with the central nervous system have been found to contain branched nerve-cells also. True neuromata form rounded or oval swellings of small size. The nerve-tubes of which they are composed are usually not continuous with those of the nerve-trunk to which they are attached, but form an irregular network mixed with a varying amount of loose connective tissue.

The most common form of **false neuroma** is composed of fibrous tissue, but myxomata, gliomata, and sarcomata are also met with. The latter differ from the fibromata in the greater rapidity of their growth, in their tendency to

infiltrate rather than simply to push aside the nerve-fibres amongst which they grow, in their greater softness, and in the gelatinous, homogeneous appearance of a freshly cut surface.

The pressure of a neuroma on the nerve-fibres amongst which it lies may cause much pain, may induce sensory or motor paralysis, or cause trophic lesions of various kinds in the peripheral parts. Neuromata are sometimes multiple, and cases have been recorded in which tumours were found on almost every nerve in the body, amounting in all to several hundreds.

The bulbous swellings which form on nerves after injury, and which are sometimes named "traumatic neuromata," are not tumours in the ordinary sense of the word, and are described in the chapter on "Injuries of Nerves."

ANGEIOMATA.

The angeiomata, or vascular tumours, are growths composed of blood-vessels, and are commonly divided into two classes—the **simple** or **capillary**, and the **cavernous** or **venous**.

The simple or **capillary angeiomata** are commonly known as "**nævi**," and are of congenital origin, although their size at birth is often minute. A simple nævus is composed of fibrous stroma containing many large and thin-walled capillaries, which frequently present irregular pouches or bulgings, and anastomose very freely. In almost all capillary nævi there are also a certain number of arteries and veins, such as are met with more especially in the cavernous variety.

Clinical characters.—This form of nævus is found most often on the face and neck, although no part of the surface of the trunk or limbs is entirely exempt. In colour cutaneous nævi vary, but are more often bright red than dusky and blue. The extent of skin implicated also differs much in different cases, but the growths are almost invariably but little raised from the surface, are soft and spongy

to the touch, and can be made to disappear almost entirely by firm pressure.

Many capillary nævi, if left alone, gradually shrink, the blood-vessels composing them being obliterated.

Cavernous or **venous angeiomata** are most often found in the skin and subcutaneous tissues, where they form "venous nævi." These tumours closely resemble in their structure the erectile tissue of the penis, being composed of a fibrous stroma enclosing large irregular cavities or spaces filled with blood, which open directly into the neighbouring veins and arteries. The cavities are lined by tessellated endothelium, and appear to be formed both by dilatation of pre-existing capillaries, with absorption of the fibrous stroma separating them, and by the development of new vessels.

Clinical characters.—The venous nævi, which involve the skin as well as the subcutaneous tissue, form irregular, bluish, or purple swellings, which are soft, and capable of being greatly diminished by pressure. When, however, they are situated entirely beneath the skin, they may cause but little discoloration. In some of these cases a considerable quantity of fat is found mixed with the cavernous tissue, and the tumour feels like an ordinary lipoma. Such growths have been named "nævoid lipomata."

CHAPTER XVIII.

MALIGNANT CONNECTIVE-TISSUE TUMOURS, OR SARCOMATA.

A sarcoma is a tumour composed of connective-tissue cells, and develops from one of the various connective tissues of the body. The type of the sarcoma cell is the connective-tissue cell, but many of the cells of the sarcomata differ from those of connective tissue both in shape and size.

In a typical sarcoma there is no definite stroma, the cells being closely packed and uniformly distributed throughout all parts of a section. Blood-vessels are numerous, the smaller arterioles having very thin walls, and often appearing as narrow channels bounded merely by the cells of the growth. No lymphatics have been demonstrated.

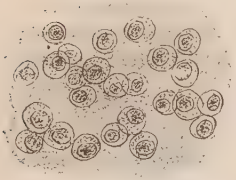
CLASSIFICATION.

The sarcomata may be divided into four chief groups: (i) **round-celled**, (ii) **spindle-celled**, (iii) **mixed-celled**, (iv) **giant-celled** or **myeloid**.

Round-celled sarcomata.—These tumours are composed of round, granular cells, with no definite cell-wall, often multinucleated, and in any individual tumour usually of the same size throughout. The cells vary much in size, however, in different growths. In some they are no larger than a leucocyte—small round-celled sarcoma; whilst in others they are more than double or treble this size—large round-celled sarcoma. The matrix is usually soft, granular or homogeneous, and abundantly supplied with vessels.

The **spindle-celled sarcomata** consist of closely-packed, oval, spindle, fusiform, or oat-shaped cells, which

FIG. 25.



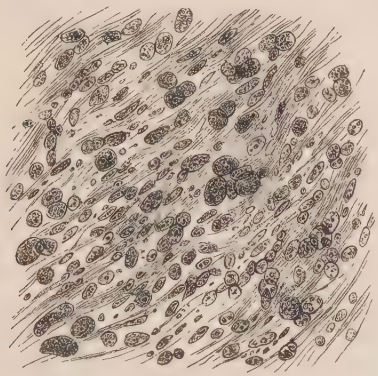
Large Round-celled Sarcoma. (Crouch : $\frac{1}{6}$ inch.)

FIG. 26.

Spindle-celled Sarcoma from the Subcutaneous Tissue. (Crouch : $\frac{1}{6}$ inch.)

are generally granular and multinucleated, have no definite cell-walls, and are mostly arranged with their long axes parallel to each other. They vary in size in different

FIG. 27.



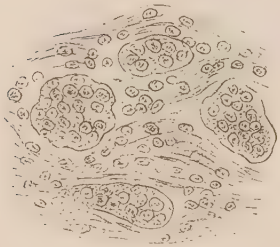
Mixed-celled Sarcoma. (Crouch : $\frac{1}{6}$ inch.)

tumours, but are usually all of about the same dimensions in any individual growth. They are collected in bundles, and are embedded in a matrix which may be either homo-

geneous or fibrillated. They are not so vascular as the round-celled variety.

The **mixed-celled sarcomata** are composed of cells of various shapes and sizes, whose general characters are similar to those of the round and spindle-celled growths.

Fig. 28.



Myeloid Sarcoma from the Lower Jaw. (Crouch: $\frac{1}{6}$ inch.)

The **giant-celled** or **myeloid sarcomata** are characterised by the presence of large multinucleated cells resembling those of the marrow of bone; these cells are often some twenty to thirty times the size of a leucocyte, and contain from ten to forty nuclei. Mingled with these are a number of oval, spindle, or round cells.

VARIETIES.

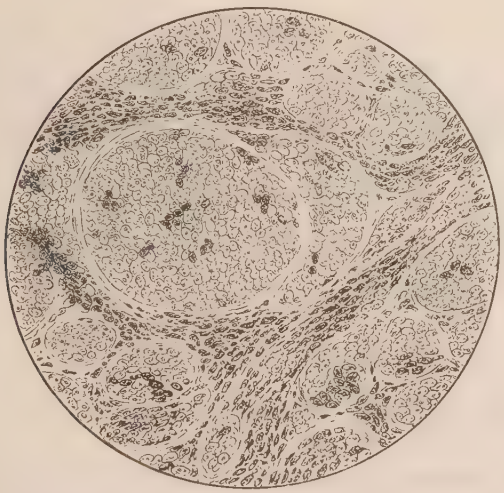
Although the sarcomata may be primarily subdivided into the above four groups, they nevertheless present numerous well-defined varieties which may now be briefly described.

Melanotic sarcomata.—These tumours are characterised by the presence of pigment, which is of a golden-brown colour when seen in minute quantities, but gives a dark-brown or black hue to the growths in which it is found. The pigment is often very irregularly distributed, and occurs both in the cells and in the intercellular matrix. Most of the melanotic sarcomata belong to the round-celled group, though some of them are composed of spindle cells; in the majority of specimens the matrix is fibrous, and forms definite alveoli.

Melanotic sarcomata originate in parts which normally contain pigment—*e.g.*, the skin and the choroid coat of the eye—and not infrequently commence in a mole or wart which has been irritated by caustics or imperfect attempts

at removal. These growths are extremely liable to become disseminated in both the viscera and the skin, and it is very noticeable that in the latter situation none of the tumours ever attain any great size, often ceasing to grow when little larger than a hazel-nut. The lymphatic glands in the neighbourhood of a melanotic growth of the skin are

FIG. 29.



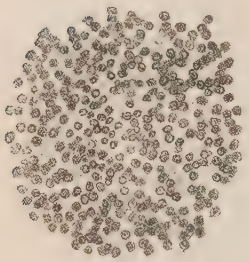
Alveolar Melanotic Sarcoma from the Skin. (Zeiss, A.)

almost always affected sooner or later by secondary deposits, and in advanced cases pigment granules may be found in the urine.

Lympho-sarcoma.—The lympho-sarcomata are composed of small round cells, about the size of leucocytes, enclosed in a fine meshwork of connective tissue whose general arrangement is precisely similar to that seen in the medullary portions of a lymphatic gland. These tumours originate in connection with lymphatic tissue, and are often seen in the lymphatic glands themselves. In many cases there is a growth of large round cells mingled with the lymphatic tissue.

Glioma and glio-sarcoma.—A glioma is a tumour composed of neuroglia, and is in many respects an innocent growth. Not infrequently however, the neuroglia tissue is mixed with much round-cell growth, and forms a glio-sarcoma, a tumour which is softer, less definitely encapsuled, and more vascular than a simple glioma. Both these

Fig. 30.



Lympho-sarcoma from the Axilla. (Crouch : $\frac{1}{6}$ inch.)

Fig. 31.

Glioma from the Cerebellum. (Crouch : $\frac{1}{6}$ inch,)

varieties occur almost exclusively in connection with the nervous system.

Hæmorrhagic sarcoma.—This is a term applied to certain forms of round- or spindle-celled growths which evince a great tendency to blood extravasations. The hæmorrhage, which is due to the extreme tenuity of the vessels, breaks up the cell masses, and forms blood-cysts of various sizes surrounded by a thin layer of the original tumour. Many of these hæmorrhagic sarcomata are liable to be mistaken for simple cysts, the amount of new growth surrounding the blood being frequently very slight. The pigmentation resulting from the disintegration of the red cells is liable to be mistaken for that of melanotic growth.

Plexiform sarcoma or cylindroma.—This is a rare variety of sarcoma, composed of round or polygonal cells, and is characterised by a hyaline degeneration of those cells which are in the immediate neighbourhood of the

blood-vessels. The unaltered cells consequently appear in section, to be contained in alveoli with hyaline walls.

Psammoma (pearl tumour).—This form of sarcoma has hitherto been found chiefly within the cranial cavity and the spinal canal. It is characterised by the presence of concentric calcareous masses surrounded by fibrous tissue and attached to fibrous trabeculæ.

Secondary changes in the sarcomata.—The most common form of degeneration is that known as "mucoid." It usually commences in the most central portions of a tumour by the distension of the cells with a clear gelatinous fluid. Gradually the whole of the cell protoplasm is liquefied, and the mucous fluid is discharged. In this way cysts are developed in the midst of the new growth, and in some cases the matrix also seems to undergo a similar change. More rarely, sarcomata undergo fatty and calcareous degeneration.

MIXED TUMOURS OF CONNECTIVE-TISSUE TYPE.

Between the sarcomata on the one hand, and the innocent connective-tissue tumours on the other, stand certain growths consisting in part of cells and in part of more fully developed tissue. The pure sarcomata are characterised by the undeveloped nature of their cells; the innocent connective-tissue tumours by the perfect development of their constituent structures. In the intermediate class are found growths which, whilst they consist in part of fully developed tissues, yet comprise many more cells than are found in the normal structures of the adult body. These growths are therefore included under the sarcomata, and a prefix is used to indicate the tissue with which the sarcomatous elements are mingled. Thus, a "fibro-sarcoma" or "fibrifying sarcoma" is a connective-tissue growth composed of a mixture of fibrous tissue and sarcoma; a "chondro-sarcoma" or "chondrifying sarcoma," a tumour of cartilage and sarcoma, &c.; and between such growths and sarcomata

on the one hand, and innocent fibromas and enchondromas on the other, no definite line can be drawn.

A fibro-sarcoma is composed of spindle cells and fibrous tissue, and all grades of development between cells and fibres can often be seen in the same specimen. These growths were formerly described as "recurrent fibroids."

A myxo-sarcoma consists of a matrix, such as has been already described under the myxomata, in which are scattered round cells.

An osteo-sarcoma is a form of sarcoma occurring in bone, in which a round or spindle-celled sarcomatous growth is developed with and in an osseous matrix; the matrix of the sarcoma becomes, in fact, converted into bone.

A chondro-sarcoma is composed of a mixture of cartilage and sarcomatous tissue combined in varying proportions.

Naked-eye appearances of sarcomata.—Sarcomata differ much from one another in appearance. The most noticeable feature of their freshly cut surface is its homogeneous, structureless appearance, and the complete absence of fibrillation or striation; in this the sarcomata differ from almost all other growths. Those tumours which are purely cellular are soft, brain-like, and pulpy, of a dirty-white colour, and very friable. Some sarcomata, especially those which are undergoing mucoid degeneration, present a gelatinous or semi-translucent appearance, whilst others are mottled by blood extravasations. In proportion as the sarcomatous tissue is mingled with fibrous, cartilaginous, or other structures, so the appearance on section of course differs. Some sarcomata present a fairly definite capsule, but the more cellular forms infiltrate in the manner common to all malignant growths. The myeloid sarcomata of bone are specially characterised by their reddish-brown or maroon colour, and their tendency to form blood-cysts.

Clinical characters of the sarcomata.—The clinical course run by the sarcomata differs not only with their

microscopical structure, but still more with the locality and tissue in which they develop. The present paragraphs, therefore, deal only very briefly with this subject, and for further information reference must be made to the chapters on the tumours of the different organs and tissues.

Sarcomata occur in the young and middle-aged rather than in the old, and occasionally follow injuries, such as contusions. The more malignant the tumour, the more rapidly it grows and tends to disseminate. Round-celled growths are the most malignant of all, and may become widely diffused and destroy life within six or eight months of their commencement. The myeloid tumours are the least malignant of the pure sarcomata, and frequently do not recur after removal. Many of the fibro-sarcomata of the skin and subcutaneous tissue also show but little malignancy, merely tending to recur locally if removed, and not affecting distant organs.

The majority of sarcomata do not affect the neighbouring lymphatic glands, though whether they do so depends rather on their position than on their microscopical structure—*e.g.*, a round-celled sarcoma of the testis almost invariably causes secondary growths in the lumbar glands, whilst a tumour of the breast of a precisely similar structure practically never causes glandular growth.

If the skin over them becomes implicated, sarcomata fungate, and form soft, bleeding masses, which discharge a mixture of blood and pus.

In some cases the structure of a sarcoma varies with its recurrence, and the recurrent growths may present a different microscopical appearance from that of the tumour which was first removed. Thus, the primary tumour may be a fibro-sarcoma with spindle cells, and the growth which recurs at the site of operation may be entirely cellular, without any fibrous stroma, and consequently of a more malignant character. On the other hand, the recurrent growth is sometimes less cellular, and therefore more innocent than the primary tumour, and, as might be expected,

the clinical course differs with the structure. Thus, the recurrent growth may grow more rapidly than the primary one, and, if again removed, may recur after a shorter interval; or the reverse may occur, and the second tumour may grow more slowly, and, after another removal, may not return until after a longer interval than that between the first operation and the first recurrence.

CHAPTER XIX.

INNOCENT EPITHELIAL TUMOURS.

Like the connective-tissue growths, the epithelial tumours may be conveniently divided into innocent and malignant, and as in the connective-tissue growths the innocent tumours are formed on the type of some one of the normal tissues of the adult body, so in the innocent epithelial tumours the adenomata and papillomata are reproductions of the normal glandular and papillomatous structures found in different parts. The carcinomata, or malignant epithelial growths, on the other hand, are formed of atypical structures which have no exact counterparts in any of the normal tissues of the adult body, and thus are strictly comparable to the malignant connective-tissue tumours—the sarcomata.

PAPILLOMATA.

The papillomata are tumours formed on the type of the papillæ which are normally developed in the skin and mucous membranes.

They consist of a stroma composed of fibrous tissue, which is in some cases richly cellular, in others dense and fibrous. In this stroma are contained both blood-vessels and lymphatics, the whole growth being frequently very vascular. The surface is covered by epithelium, which varies in its nature according to the locality of the growth. In papillomata on the skin the epithelial cells are squamous, and are arranged in several layers, whilst in growths from the mucous surface of the bladder or intestine the cells are columnar and not so thickly placed.

The fibrous stroma extends towards the free surface papillary processes, which in the "simple" papillomata are single and undivided, but in the "compound" papillomata are branched, giving off processes, which in their turn again bifurcate. The surface of a papilloma is generally irregular and villous, but is occasionally smooth, presenting no signs of subdivision of the stroma into papillæ.

Secondary changes are common in these growths, for their exposure on free surfaces renders them peculiarly

FIG. 32.

Papilloma from the Anus. (Crouch : 1 inch.)

liable to injury, and thus ulceration and hæmorrhage are of frequent occurrence.

Clinically, the papillomata are entirely innocent, and must be clearly separated from malignant tumours, such as epitheliomata, whose surfaces are also often covered by papillæ, but which differ structurally in the most essential details from simple papillary growths.

Papillomata are met with on the skin as warts and corns, and on the mucous membranes of the nares, tongue, bladder, larynx, and intestines.

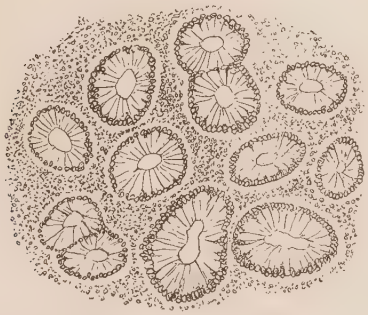
In syphilis also, and in cases of gonorrhœa, mucous tubercles or condylomata, whose structure is essentially papillomatous, are of common occurrence.

ADENOMATA.

The adenomata or glandular tumours are growths composed of gland tissue more or less perfectly developed, and are formed on the pattern of the normal gland tissue found in the different parts of the body from which they spring.

Putting aside the lymphadenomata, which are described elsewhere, and the glandular hypertrophies, which are not to be included amongst the true tumours, the adenomata may be said to consist of acini and ducts when developing

FIG. 33.



Tubular Adenoma of the Rectum. (Crouch : $\frac{1}{2}$ inch.)

in a racemose gland, or of tubes when growing from the tubular glands. They differ from the malignant epithelial growths or the "atypical epithelial tumours" in that, whilst the latter are not formed after the type of the normal epithelial tissue from which they spring, the true adenomata are exact or close imitations of glandular structure, the tubes and acini forming them having a definite lumen, and their epithelium being sharply defined from the surrounding tissue. The malignant epithelial growths, again, are not encapsuled, but infiltrate the tissues amongst which they grow, whilst the adenomata do not infiltrate and are generally encapsuled. Although composed of gland tissue, the

latter in the adenomata is frequently not physiologically active, and its secretion, if formed, is not discharged into the ducts of the gland in which it grows. In consequence of this the tubes and acini are extremely liable to become distended and to form cysts.

The adenomata of the different glands are described in subsequent chapters.

CHAPTER XX.

MALIGNANT EPITHELIAL TUMOURS, OR CARCINOMATA.

The carcinomata are tumours growing from, and composed of, epithelial cells, which are generally contained in spaces or alveoli whose walls are formed of fibrous tissue. Within the alveoli the cells are not separated from one another by any stroma or matrix, and, whilst varying in shape and size, closely resemble in their general characters the epithelial cells of the part from which they spring. They are frequently multinucleated, and increase both by fission and by endogenous cell formation. The matrix is vascular, and blood-vessels run into the alveolar walls, though they do not penetrate into the alveoli or run amongst the cells, as do the vessels of the sarcomata. Lymphatics are well developed and accompany the vessels.

The following are the chief groups of the carcinomata :—

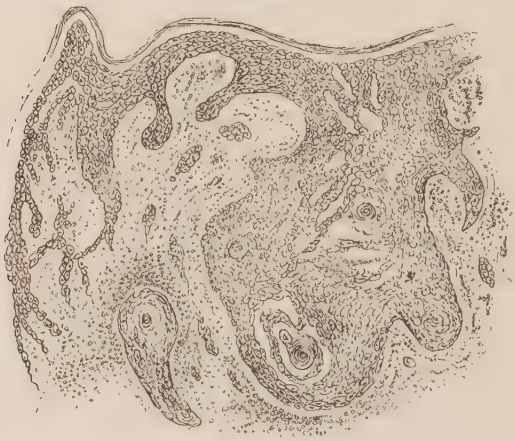
1. **Epithelioma, or squamous-celled carcinoma.**
2. **Rodent ulcer.**
3. **Spheroidal-celled carcinoma**—
   (*a*) Scirrhous.
   (*b*) Encephaloid.
   (*c*) Colloid.
4. **Cylindrical-celled carcinoma** ; adenoid cancer ; adeno-carcinoma.

EPITHELIOMA.

An epithelioma consists of a new growth of squamous epithelial cells, which not only grow, as in the papillomata,

towards the free surface, but also infiltrate and destroy the subjacent tissues. Microscopically, these tumours are found to originate in growths of solid columns of cells from the rete mucosum of the inter-papillary processes which are common to all cutaneous and mucous surfaces. As these ingrowths extend into the subjacent tissues they throw out lateral branches, which, uniting with similar offshoots from

Fig. 34.



Epithelioma of the Lip. The epithelium has grown into the subjacent tissues, and has caused a considerable exudation of cells in its neighbourhood. (Zeiss, A.)

other columns, form an irregular epithelial network. At the same time that this ingrowth occurs, an outgrowth also takes place, and a warty excrescence of epithelium develops, the superficial cells of which soon break away, and thus expose the deeper parts of the growth. In this way a malignant ulcer is developed, whose base and edges are alike formed by the tumour. This disintegration of the superficial cells continues with a rapidity equal to that of their development, and thus, whilst the new growth constantly extends amongst and destroys the subjacent structures, the

ulceration generally prevents the tumour from attaining a considerable size. Around the ingrowing columns there is generally evidence of irritation of the tissues into which the epithelioma is making its way, for the cells of the new growth behave as foreign bodies in respect to the other tissues, and the growing edge of each column is thus surrounded by much small-cell infiltration.

FIG. 35.

Cell-nests from an Epithelioma, showing the concentric arrangement and semilunar shape of the external layers of cells. (Crouch : $\frac{1}{6}$ inch.)

The epithelial cells of which the growth is composed are usually large, very irregular in shape, often possessing processes, multinucleated, and sometimes in the process of proliferating by endogenous cell-formation. Embedded in the epithelial columns are **cell-nests**, which vary much in number and size in different specimens. They are composed of central cells of a rounded shape, which in some

specimens are much broken down and degenerated. Around these are small crescent-shaped cells with their concavities inwards, and outside these again are layers on layers of cells, similar in shape, but of larger size. The nests are formed by the very rapid proliferation of cells at various foci in the tumour. The cells first formed at any focus are pushed towards the periphery by the constant and rapid growth in the centre, and become flattened or crescentic through the resistance offered by the surrounding tissues. The chief significance of a nest is that it indicates rapid cell-growth, and therefore, when occurring in the deeper parts of the skin, or in the subcutaneous tissues, is diagnostic of epithelioma, for no such formations exist under normal circumstances in these situations.

Locality.—The most common situations for epitheliomata are the junctions of mucous and cutaneous surfaces. They occur on the lips (usually the lower lip) and other parts of the face, the tongue, the larynx, the œsophagus, and the anus; on the penis, the scrotum, the vulva, the os uteri; more rarely on the skin of the extremities or trunk, especially in the site of old scars, ulcers, or sinuses, and in the bladder.

Clinical characters.—Epithelioma is essentially a disease of late life, and is seldom seen before the age of forty. It commences as a warty or papillated growth, which increases rapidly and infiltrates the tissues amongst which it develops. The tumour ulcerates early, the ulcer having a foul, sloughy base and raised indurated edges formed of that part of the epithelioma which has not yet become involved in the ulcerative process. The destruction of tissue is at times very great, and is accompanied by much pain. The neighbouring lymphatic glands are early affected, and in them the epithelioma runs a course precisely similar to that of the primary tumour. The masses of growth in the glands commonly attain a much greater size than does the original tumour, and not infrequently suppurate. Secondary growths in the viscera are not common, although

they do occur, and death generally results from direct extension to important structures, and from exhaustion caused by pain and by discharge of blood and pus.

It should be mentioned that, in addition to the squamous-celled carcinomata, epitheliomata composed of columnar cells grow from the mucous membrane of the nose and antrum.

Cystic Epithelioma is a rare variety of the disease which occurs in the neck. These growths almost certainly originate from the epithelium of one of the branchial clefts, and are characterised by their deeply-seated situation and by the presence of large collections of blood-stained serum in their interiors. They simulate abscesses and run a rapid course.

RODENT ULCER.

A rodent ulcer is a tumour composed of epithelial cells, and is developed in the derma, apparently from the

FIG. 36.

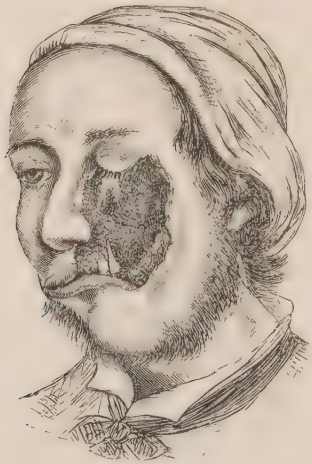
Rodent Ulcer from the Face. The surface epithelium has not proliferated, and, beneath it, the derma and subcutaneous tissue are infiltrated with an irregular growth of small round and oval cells. (Crouch : ½ inch.)

glandular structures, such as the sebaceous glands, or the hair follicles, which are found in this situation. It thus

differs in its origin from epithelioma, which is essentially an ingrowth of the surface epithelium. The exact site of its origin is still not definitely settled, but it is commonly believed to spring from the sebaceous glands, although some pathologists refer its development to the hair follicles. There is no proof that it originates in the sweat glands.

The cell-growth invades the tissues in the form of large flask-shaped or irregular ingrowths, frequently separated

FIG. 37.



Rodent Ulcer with sharp-cut edges and very little new growth.

from each other by several normal papillæ and interpapillary processes. The cells themselves are small and round, being usually not more than one-third as large as those forming the epitheliomata, and, unlike the latter, they do not become horny or show any tendency to keratinoid changes. They show a great tendency to become vacuolated, and thus form spaces in the centre of the ingrowing mass. In most specimens there are no cell-nests, and these, when present, are very small, ill-developed, and sparsely scattered. The epidermis is infiltrated from below, and being destroyed

after a time, allows of the protrusion of the cell-growth, an event which is followed by ulceration or disintegration, as in the case of epithelioma.

Locality.—Rodent ulcer is almost limited to the face, and is most common on the side of the nose, at the inner angle of the orbit, on the forehead, and the prominence of the malar bone. It is rare on the lips and chin. It never

FIG. 38.

Rodent Ulcer with raised, rounded edges, and a considerable amount of new growth.

occurs on the extremities, but occasionally grows on the neck and trunk.

Clinical characters.—Rodent ulcer is seldom seen before the age of thirty-five or forty, but may develop as early as the period of puberty. It commences as a small pimple covered by smooth thinned epidermis, which is never papillated like a wart, and the centre of which, in time, ulcerates; the growth of the pimple and the spread of the ulceration progress with about equal rapidity, so that at no time is there any large amount of new growth. Frequently the ulceration heals in one place whilst it extends in another; but the scar which forms is not stable, and soon again breaks down.

The growth of these tumours is essentially slow, though the rate of growth differs somewhat in different cases. An ulcer as large as a sixpence is often found to be a year or more old. When fully developed, a rodent ulcer is a flattened growth of an irregular shape, with a grey, smooth, and glazed base, devoid of healthy granulations, and discharging thin watery pus. In parts a little ill-formed scar tissue may be seen, and the surface is often covered by a scab. The edges are sometimes sharply cut and not raised, but more frequently are both raised and indurated. They are never papillated or warty like the edges of many epitheliomata, but, even when considerably raised, present a smooth, rolled appearance, the epithelium covering them being thinned and bluish. The lymphatic glands are never affected by secondary tumours, and the viscera remain throughout free from disseminated growths. Although large portions of the face are commonly destroyed, rodent ulcer is so slow in its progress that the patient often dies from old age or intercurrent disease before the tumour reaches a vital part, such as the brain. In some cases the whole of the lips, nose, eyes, and mouth are destroyed before death occurs.

SPHEROIDAL-CELLED CARCINOMA.

The spheroidal-celled carcinomata are subdivided into three groups: (1) Scirrhous; (2) Encephaloid; (3) Colloid.

Scirrhous carcinoma.—To the naked eye a scirrhous carcinoma appears as a white, fibrous mass very firm and dense, cutting with a peculiar grating or creaking sensation, concave on section, and with an irregular margin. The tumour is not encapsuled, and at its edge may be seen small masses of fat, or other normal tissues which are being infiltrated by the growth, and which have, as it were, been entangled by it, preparatory to being absorbed. The soft parts around the cancerous growth are puckered and drawn towards the tumour by the contraction of the branching processes which extend from the margins of the latter.

Examined microscopically, a scirrhous carcinoma is found to consist of spheroidal epithelial cells lying in a stroma composed of interlacing bands of fibres, and containing a varying proportion of connective-tissue cells. The alveoli or spaces, enclosed by these bands are very irregular in shape and size, and are smaller in the more central parts of the growth than at its periphery. The epithelial cells, though generally spheroidal, vary a good deal in shape and size, being larger and more fully developed where the alveoli are

FIG. 30.

Scirrhous Carcinoma of the Breast. (Crouch : $\frac{1}{6}$ inch.)

large, whilst in the most central parts of the growth they may often be seen to contain fatty granules. Some of the alveoli contain as many as thirty or forty cells or more, although in other portions of the growth the latter lie in groups of but two or three, or in single file amidst the bundles of connective tissue.

Secondary changes in scirrhous carcinomata are of common occurrence, for, in addition to ulcerating when they reach a surface, they are liable to undergo fatty degeneration in their more central parts or to become the seat of blood extravasations and cysts. Most rarely suppuration occurs, and pus is formed in the substance of the tumour.

Locality.—By far the most common seat of scirrhous carcinoma is the female breast, but this variety of cancer is also found in the uterus, œsophagus, stomach, prostate, and skin.

Clinical characters.—Scirrhous cancer is most common in women after the age of forty. It commences as a firm knotty lump which is at first painless, and may thus attain a considerable size before attracting attention. When fully developed the surface of the tumour is irregular, nodular, and very hard, sometimes feeling like a mass of cartilage. Though at first movable, the growth soon contracts adhesions, and causes puckering and retraction of the skin and adjacent parts. The rapidity of growth varies much in different localities, and in the same locality in different patients. After a variable time the skin or mucous membrane covering the tumour becomes involved, and a sloughing mass protrudes, from which both pus and blood are discharged. The lymphatic glands in the neighbourhood are early affected by secondary deposits, and, as time goes on, the thoracic and abdominal viscera, and more rarely the bones, become the seats of disseminated growths.

Encephaloid carcinoma.—This variety of the spheroidal-celled cancers is of much more rare occurrence than the scirrhous growths. To the naked eye an encephaloid carcinoma presents a soft, greyish or dirty-white, brain-like mass, mottled in places by hæmorrhages, frequently containing cysts, and readily reduced to a pulp by slight pressure. It is nowhere encapsuled.

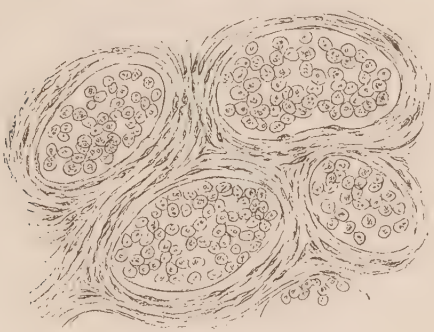
Microscopically examined, an encephaloid carcinoma consists of a fibrous stroma enclosing alveoli in which are contained spheroidal epithelial cells. The alveoli are larger and more regular in size than in scirrhous cancer, and their walls are much more delicate, being composed of finely fibrillated connective tissue instead of the dense fibrous stroma of the scirrhous tumours. The epithelial cells contained in the alveoli constitute by far the greater portion of the growth. They are large and round, with definite nuclei,

and well-developed cell-bodies; they often contain much granular matter and many nucleoli.

Locality.—The female breast, testis, stomach, bladder, and superior maxilla are the parts of the body in which this form of tumour is found.

Clinical characters.—Medullary cancers are soft, elastic, or semi-fluctuating swellings, of extremely rapid growth, quickly involving the skin, readily fungating, causing early implication of the glands, and disseminating

FIG. 40.



Encephaloid Carcinoma of the Antrum. (Crouch : $\frac{1}{6}$ inch.)

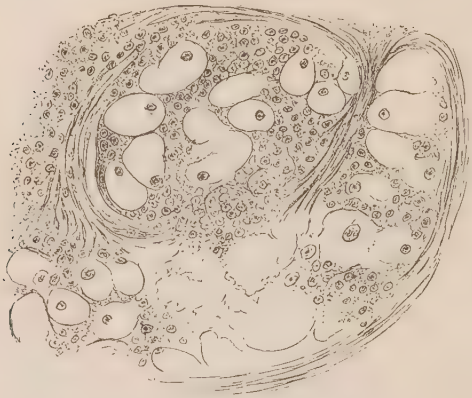
in the viscera with great rapidity. In many cases life is destroyed within a year of the first appearance of the original tumour. As many of the round-celled sarcomata were formerly included under the medullary cancers, these latter growths are generally supposed to be much more common than is really the case. Their occurrence is indeed of considerable rarity.

Colloid cancer.—Colloid cancer is merely a variety of scirrhous or encephaloid carcinoma in which the cells, and in many cases the stroma, undergo colloid degeneration. To the naked eye a colloid carcinoma presents an irregular network, the spaces of which are filled by a clear, jelly-like, or slightly opalescent, gelatinous substance. The network

is most open and the colloid matter most abundant in the central or oldest portions of the tumour, whilst the periphery is much like that of a scirrhous growth in its irregular, infiltrating edge, and nodulated surface.

Microscopically examined, a section shows the alveolation of the matrix and the masses of epithelial cells common to the other varieties of carcinoma. Some of the cells are of the same size and shape as those of the glandular tissue

FIG. 41.



Section of a Carcinoma of the Breast. Many of the cells are distended with colloid matter, and in the lower part of the section is an irregular cavity caused by the bursting of some such cells. (Crouch : $\frac{1}{6}$ inch.)

from which the growth springs, but others are filled in varying degrees with colloid matter which thrusts the nucleus to one side and distends the cell-body until it bursts. By a continuance of this process in the other cells, they are gradually destroyed, and their place taken by the colloid substance which now fills the alveolus. The remains of the walls of the ruptured cells can often be seen after their contents have been discharged. In many cases the fibrous matrix also undergoes degenerative change, and the fibres composing it swell up with colloid matter, whilst their

outlines become blurred and indistinct. As the walls of the alveoli are thus destroyed, the cavities of those contiguous to one another become continuous, and thus in the most central or oldest portions of the tumour, as already stated, the network is more open than at the edges.

Locality.—Colloid carcinomata are most common in the stomach, but are found also in the breast and intestines, and more rarely in other parts of the body.

In their **clinical characters** these tumours do not materially differ from the scirrhous cancers, although they are not generally so malignant, growing more slowly and seldom affecting the viscera; they require no separate description.

The cylindrical-celled carcinomata are fully described in the chapter on "Tumours of the Rectum." They occur also in the stomach and small intestine.

CHAPTER XXI.

CYSTS.

A CYST is a closed sac with fluid or semi-fluid contents. The following groups are generally recognised :—

1. Cysts formed by distension of pre-existing tubes or cavities.
2. Cysts of new formation.
3. Cysts of congenital origin.

The cysts formed by distension of pre-existing tubes or cavities are the most common, and are developed in different ways. Thus, mucous cysts are the result of some obstruction to the ducts of the mucous glands, and sebaceous cysts to a similar retention of the products of the sebaceous glands, whilst cysts in the breasts or kidneys result from retention of the mammary or renal secretion. In other cases, again, the cysts are developed by distension of a cavity which normally has no duct or outlet. Thus, cysts of the thyroid gland, enlarged bursæ, vaginal hydroceles, &c., are the result of an excessive secretion of fluid from the epithelial or endothelial cells of the respective parts.

Sebaceous cysts.—These are formed by blocking of the duct of a sebaceous gland, most often by dirt, or by surrounding inflammation. They are most common on the scalp, face, neck, and shoulders, and are seen at all ages after childhood. In size they vary from that of a pea to that of a hen's egg. They are situated in the substance of the skin, and the latter, in consequence, cannot be moved

freely over the swelling, whilst on the most central portion of the tumour, a small black speck, which is the obstructed sebaceous duct, can often be distinguished. When large, the cyst extends into the subcutaneous tissue.

A sebaceous cyst contains sebaceous matter, the wall of the cyst being formed by the epithelial cells of the secreting portion of the gland, arranged in numerous layers, and placed upon a basement membrane of fibrous tissue.

In some cases sebaceous cysts become inflamed and suppurate, and thus give rise to abscesses or ulcers which are generally very slow to heal.

Mucous cysts occur in connection with the glands of mucous membrane, as do the sebaceous cysts in connection with the glands of the skin. They are formed by distension of the mucous glands with secretion. They may occur on any mucous surface, and are commonly seen on the lips, the tongue, and the vulva.

Cysts of new formation develop in different ways. Some result from blood extravasations, as do the so-called "apoplectic cysts" of the brain and the arachnoid cysts; others, like the adventitious bursæ, result from the accumulation of fluid in the lymphatic spaces of the connective tissue, and are commonly known as **serous cysts**, whilst a third class are formed in connection with foreign bodies (*e.g.*, bullets).

Hydatid cysts result from the presence of a parasite in the tissue. The parasite in question is the larval form of the Tænia echinococcus, a small tape-worm found in dogs and wolves. The eggs of this worm are passed in the fæces of the animal, and obtain access to the human body through the ingestion of impure water, or of watercress and other such uncooked vegetable matter. The membrane which surrounds the egg is dissolved in the secretions of the stomach or intestines, and, the embryo being freed, passes with the venous blood or the lymph to other parts of the body. The liver is more often affected by hydatids than

any other organ, probably for the reason that so much of the blood from the alimentary canal passes directly through it, but hydatid cysts may occur in any tissue or organ.

The immediate result of the lodgment of an embryo echinococcus is such an amount of irritation of the surrounding connective tissue that cell-exudation and the formation of fibrous tissue soon cause the parasite to be enclosed in a fibrous wall or envelope, which in all cases constitutes the outermost layer of an hydatid cyst. Within this capsule lies the true cyst-wall, in which two layers may be distinguished. The outer of these is composed of a homogeneous, elastic, laminated membrane of appreciable thickness, to which the name of *Ectocyst* has been applied by Huxley. It is very vascular, and its vessels are commonly somewhat varicose. Within this is the *Endocyst*, a thin, granulated, non-elastic membrane, from which are produced buds, which afterwards develop into echinococcus heads or scolices. Other and smaller cysts—daughter cysts—also develop from the lining membrane, and themselves also produce scolices.

Each scolex is about 0·3 mm. in length, but the fore-part of the body is not infrequently found withdrawn or invaginated into the hinder-part. In its parenchyma are numerous round or oval calcareous particles, and its most prominent anterior part or rostellum contains a ring of calcareous hooklets. The hydatid fluid which distends the mother and daughter cysts alike is clear and watery; does not coagulate on being heated; has a specific gravity varying from 1005 to 1012; is neutral or alkaline in reaction, and contains chloride of sodium, but no albumen. Examined under the microscope, the fluid is often found to contain some of the above-mentioned calcareous hooklets.

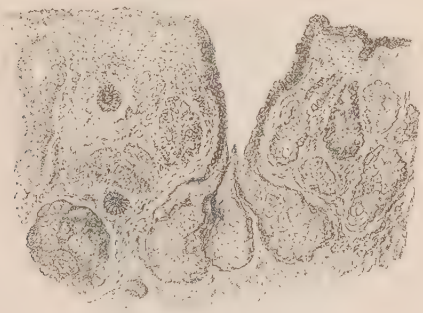
If left alone, an hydatid cyst may increase in size and finally rupture into the surrounding tissues, may suppurate or, the fluid being absorbed, the contents may become converted into a cheesy or putty-like mass, and the

cyst-wall be rolled up on itself and remain encapsuled in the tissues. In some cases also hæmorrhage occurs, and the blood undergoing degenerative changes, the cyst may be found filled with chocolate-coloured fluid, the parasite having died.

Cysts of congenital origin appear to be formed by inclusion of portions of epiblast or mesoblast during the process of closure of the thoracic, abdominal, branchial, or other cavities.

Dermoid cysts have a cyst-wall formed of true skin, with papillæ, hair follicles, sudoriparous and sebaceous

FIG. 42.



Section of the Wall of a Dermoid Cyst of the Ovary, showing large sebaceous glands and cross sections of hairs. (Crouch : $\frac{1}{2}$ inch)

glands. They are always of congenital origin, but frequently do not become noticeable before childhood or puberty. They contain sebaceous matter, and increase in size by the constant secretion of the sebaceous glands which line the cyst-wall, and empty their contents into its cavity. More rarely they contain bone, teeth, &c.—a circumstance which is generally accounted for by the supposed inclusion of portions of mesoblast as well as of epiblast.

Dermoid cysts are common at the angles of the orbit and in the eyelids, where they are always found beneath the

deep fascia, the skin being quite movable over them. They also occur with some frequency in the ovary, and more rarely in the floor of the mouth, the middle line of the neck, and the testis.

In the neck, dermoid cysts are also met with in the lines of the branchial clefts, and are here called "branchial cysts." They are formed in this situation from the epiblastic tissues lining the clefts, and are always found beneath the deep fascia. In the lines of the branchial clefts also small cartilaginous growths may develop. They have been called "accessory auricles," and are composed of irregular

FIG. 43.

masses of hyaline cartilage, with a little fibrous tissue. They are often associated with branchial cysts. (See Fig. 43.)

Further description of dermoid and other congenital cysts will be found in the chapters devoted to particular regions.

Cystic hygroma.—This name is applied to certain cysts of congenital origin, which are most often met with in the neck and the axilla; they are also named "hydrocele of the neck."

These growths are generally seen in young children, in whom they form tense and painless swellings of various sizes. In some cases they grow rapidly; in others they shrink and dwindle away. In yet other cases they persist, but do not alter in size.

Dissection shows either single or multiple cysts, generally situated in the submaxillary region, the axilla, or the posterior triangle of the neck. The cysts are formed by a delicate wall of connective tissue, with a smooth lining membrane, enclosing clear serous fluid, and in some cases are not surrounded by any solid new growth.

More often, however, around and between the cysts is some soft fibrous tissue, very gelatinous and semi-translucent, and not unlike Wharton's jelly in a recently cut umbilical cord. The hygromata are in fact formed from portions of included mesoblast, and are composed of a loose connective tissue whose spaces are distended with fluid.

In solid tumours, also, **degeneration cysts** are liable to form, especially in the sarcomata, enchondromata, and soft fibromata. These, however, are not to be included amongst the true cystic tumours.

CHAPTER XXII.

CONTUSIONS.

A CONTUSION is a subcutaneous, lacerated wound. The various soft tissues are torn, and, the vessels being injured, blood escapes and stains the neighbouring parts. The damaged structures are repaired by a process of plastic inflammation with organisation of the inflammatory products into fibrous tissue ; suppuration is rare, but occasionally occurs.

The amount of the subcutaneous hæmorrhage which accompanies a contusion depends on the extent of the injury, the resistance offered by the tissues to the extravasated fluid, and the size of the injured vessels. In cases where a large area of tissue, not containing any important vessels, has been the seat of contusion, the hæmorrhage will be diffuse, and will result in the production of a general **ecchymosis,** but where the injury is more localised, where the tissues are lax and yielding, or where a large vessel has been torn, the effused blood is collected into a mass, and forms a circumscribed extravasation, a blood-tumour, or a **hæmatoma.**

In the case of a general ecchymosis the following changes occur in the extravasated fluid :—The blood clots, and the fibrin causes a certain amount of induration. It is subsequently either replaced by a growth of new fibrous tissue, or, more probably, is absorbed by the leucocytes, which are soon exuded as the result of the injury. The serum and

leucocytes escape into the neighbouring lymphatics, and are thus removed. The red blood-cells disintegrate, and their colouring matter is diffused amongst, and stains, the surrounding parts. The hæmoglobin undergoes a series of changes, which finally result in the production of hæmatoidin, and the colour of the contused parts passes through various hues, the last being a pale lemon or citron. Crystals of hæmotoidin of this latter tint may be found for many months in the neighbourhood of a contused part.

The removal of the blood which forms a "hæmatoma" requires much more time than the disintegration of a simple ecchymosis. The coagulated fibrin surrounds the fluid portion of the blood, and is itself surrounded by newly formed fibrous tissue, which results from the irritation of the neighbouring parts by the mass of extravasated blood. New vessels are formed, and penetrate the dense wall which encloses the serum and corpuscles, and after a time the latter are slowly absorbed. The red blood-cells disintegrate in the manner already described, but their colouring matter, instead of staining the tissues, as in a general ecchymosis, is diffused throughout the serum in which the cells are suspended, and thus the fluid contents of the hæmatoma become generally stained, and are often of a dark chocolate or tarry colour. If the serum is now absorbed, the fluid contents of the blood-cyst become thickened and inspissated, but if absorption does not occur, then the hæmoglobin gradually undergoes a series of changes which result in its loss of colour, the formation of hæmatoidin, and the production of a cyst which may contain an almost clear fluid. Such a condition as this is more often seen in hæmorrhages into the brain or the arachnoid cavity than elsewhere.

In other cases, again, the swelling caused by the blood-tumour never subsides, and a permanent hard lump may persist, which on section is found to be composed of fibrous tissue. The blood-clot is said to have become organised but the exact manner in which this organisation takes place

does not differ from that about to be described as occurring in the internal coagula of injured vessels.

Lastly, where the parts have been so injured that suppuration ensues, the extravasated blood tends to decompose. It becomes mingled with the products of suppuration, and together with them is discharged.

CHAPTER XXIII.

HÆMORRHAGE AND INJURIES OF VESSELS.

Hæmorrhage is the escape of blood from the vessels within which it should normally remain. The escape may be due to either disease or injury, but it is with the latter alone that we are at present concerned.

If a wound be inflicted on almost any part of the body bleeding results, but in the very large majority of cases the flow of blood is arrested without recourse to any artificial means, and it is only when some large vessel is opened or the patient is a "bleeder" that death ensues from loss of blood or syncope.

It is well known that blood tends to coagulate when no longer in contact with the endothelium of a healthy vessel, and, where nothing but small capillaries and arterioles have been wounded, the coagulation of the blood is alone sufficient to plug the apertures in the vessels, and to arrest the flow. But where larger arteries have been wounded, and the loss of blood is great, changes occur in the injured vessels themselves which help to bring about what is always the final means of stoppage, namely, the coagulation of the blood.

If an artery be cut across, it immediately **retracts** by virtue of its elasticity, and, by thus retracting, its bleeding orifice tends to be covered up and compressed by the tissues amongst which it is placed; the flow of blood is thereby diminished, and coagulation is favoured. Another effect of injury to a vessel is **contraction.** This is not the result

of elasticity, but of muscular action which is brought into play by both the injury and the exposure of the muscle-coat. So greatly is the lumen of a vessel diminished by this means, that, in small arteries, the blood-flow may be entirely arrested by it alone.

It has already been said that the whole artery retracts within the surrounding tissues, but, in addition, the internal and middle coats, which are more elastic than the outer, retract yet more, and by curling in towards the lumen, still further arrest the blood-stream.

Lastly, the more blood the patient loses the more coagulable does the remaining blood become, and the weaker is the heart's action and the blood-stream.

In these various ways, therefore, the blood-stream is weakened in cases of wounds of large vessels, but it must be distinctly borne in mind that each and all tend to one end, namely, **the coagulation of the blood.** If the latter did not occur, all the other events would be useless, and it is by the formation of a clot in the tissues outside the wounded vessels, the so-called "**external coagulum**," that the bleeding is **temporarily** arrested.

This external clot is, then, but a temporary expedient; it is placed at the end of the bleeding vessel until a sufficient amount of time has elapsed for the artery to be permanently sealed, and, by the time this result has been attained, the external coagulum is undergoing absorption, and is subsequently entirely removed.

The **permanent** occlusion of the injured vessel is accomplished by a process of plastic inflammation, *i.e.*, by an exudation of inflammatory products, and a subsequent organisation of the latter into fibrous tissue. Soon after the external clot has been formed and bleeding has ceased, the ordinary phenomena of inflammation make their appearance at the injured part, the cut ends of the injured vessel as well as the surrounding tissues become swollen and more vascular, exudation of inflammatory products results, and within a few hours the end of the cut vessel may be

found embedded in a small mass of lymph. This lymph very shortly becomes vascularised by the formation of new blood-vessels from the vasa vasorum of the injured artery, and finally undergoes organisation into fibrous tissue in a manner precisely similar to that which occurs in the healing of a wound by first intention. In this way the coats of the artery become matted to one another and to the sheath, and the whole end of the cut vessel is plugged by a firm mass of fibrous tissue continuous with the scar tissue of the wounded cutaneous surface.

At the same time that these changes are in progress, the blood in the injured vessel also clots, and the clotting extends a little way up from the seat of injury, never reaching beyond the origin of the first large branch given off above the wound. This clot, in contradistinction to that outside the vessel, is called the **internal coagulum**, and much more importance has been attached to it as a means of permanent occlusion than it deserves. The internal coagulum owes its origin to either the stopping of the blood-stream or to the proximity of the inflammatory exudation, for blood always tends to clot where the tissues are inflamed. The following changes occur in the clot after its formation :—

At first it is of a dark-red colour, and not at all adherent to the vessel-wall. In a few days it becomes of a lighter hue, and adherent to the artery; gradually it becomes white and firm, and in the process shrivels in all its diameters. Finally, it becomes in part detached from the walls of the artery, and remains attached by its base alone to the fibrous tissue, which by this time plugs the end of the vessel.

If sections be made of the clot and of the artery which contains it during this process of organisation, the following changes will be observed :—

At first the section shows nothing but red blood-cells entangled in a network of fibrin. Within a day or two these red blood-cells have all disintegrated, and connective-

tissue cells in greatly increased numbers are now seen. By the third or fourth day newly formed vessels penetrate into the clot from the vascularised lymph at the seat of injury, and supply the cell-growth with nourishment. By the eighth or tenth day the whole clot is formed of new cells, which have displaced and destroyed the original clot and its red blood-cells, and are already undergoing organisation into fibrous tissue. The newly formed vessels now shrink and disappear, the young fibrous tissue of the clot also shrinks, and, finally, a firm fibrous plug is all that remains of the internal coagulum.

Of what use, then, is the internal clot? It acts as a buffer, and prevents the rush of blood, which would otherwise tend to wash away the exuded lymph from the cut end of the vessel. It further tends to act as a permanent plug, and although of itself it would be quite insufficient to stop the forcible blood-stream in a large vessel, it is yet of some service in this respect.

CONTUSION OF ARTERIES.

Contusions of arteries may either set up inflammatory changes in the vessel without any definite injury being produced, or may cause a laceration of the internal and middle coats without tearing the external coat or the sheath. In the latter case the effects of the contusion are precisely the same as those produced by the application of a ligature, and the inner coats alone give way, on account of their more brittle nature. The torn coats curl in towards the lumen, and obstruct the blood-stream. Very soon after the injury, coagulation of the blood commences at the injured spot, and the artery is gradually occluded both by the coagulated blood, and later on by the formation of fibrous tissue produced for the repair of the damaged vessel. The condition of the limb below the seat of injury is exactly the same as it would be after ligature—cold, white and pulse less. If the collateral circulation be sufficient to keep it alive, all will go well; if not, gangrene will ensue. Un-

fortunately, however, cases of partial laceration of an artery are seldom uncomplicated. The injury which damages the vessel is very likely to implicate its accompanying veins, and, if the circulation in the latter be also arrested, the chance of the occurrence of gangrene is greatly increased.

RUPTURE OF ARTERIES.

Complete subcutaneous rupture of a large artery is an accident of rare occurrence, and is more frequently seen in the popliteal and brachial than in any other vessel. The immediate result of complete rupture is the rapid extravasation of blood into the neighbouring soft tissues. Forcibly driven by the heart's action, the blood may be propelled to considerable distances along the fascial planes, and the whole limb becomes livid, swollen, pulseless, and cold. At first the swelling situated over the injured vessel pulsates, but as the parts become more and more filled with coagulated blood, it is impossible for the heart to distend them further, and pulsation ceases. If nothing be done, gangrene will ensue, not only because the arterial flow is interfered with, but also because the extravasated blood causes pressure on the veins, and so prevents the return of the venous blood. It is of the utmost practical importance to recognise this latter fact, for if any attempt were made to treat the case by ligature of the artery above, as though it were one in which the effused blood was in a definite sac, gangrene would inevitably ensue.

TRAUMATIC ANEURYSM.

A traumatic aneurysm is a "tumour containing blood communicating with the cavity of an artery," and resulting from an injury.

Traumatic aneurysms may be caused either by open wounds or by subcutaneous lacerations of an artery. In the former case the vessel is usually opened by a puncture with a sharp instrument, which is at once withdrawn;

hæmorrhage immediately ensues, but is arrested by pressure. The pressure, although sufficient to prevent the egress of blood from the skin-wound, does not suffice to prevent it from slowly leaking out of the wounded artery. A collection of blood is thus formed in the surrounding tissues, and these, being irritated by its presence, become matted and condensed by fibrous tissue. By this condensation, the further extravasation of blood is prevented, and an "aneurysmal sac" is formed, which soon becomes thickened and strengthened by coagulation of some of the blood within it. It is immaterial whether the original hæmorrhage be arrested by artificial pressure or by the resistance offered to the escape of blood by the muscles and fasciæ through which it flows.

In other cases an artery is subcutaneously torn or otherwise injured without any skin-wound being caused—*e.g.*, in fractures or dislocations, as well as, more rarely, in simple contusions. Under these circumstances, as in punctured wounds, the resistance offered by the surrounding parts is often sufficient to prevent a wide extravasation of the blood, and the latter becomes limited by condensation of the soft parts, and by clot, in the same way as in the former case.

Another mode of formation of traumatic aneurysm has been described, but is of somewhat doubtful occurrence. In punctured wounds of arteries which have healed, the resulting scar is supposed to become distended by the blood-pressure, and an aneurysmal sac is thus formed by dilatation of the vessel at the seat of injury.

The limitation of the flow of blood by the formation of the sac is to be regarded as the first step towards the healing of the injured artery. If no sac were formed, gangrene would ensue, but, the escape of blood having once been limited, the tendency to a natural cure is very considerable, and any aid afforded by rest and artificial pressure is most likely to bring about a gradual filling up of the sac by the deposit of laminated fibrin from the flowing blood, and to result in

the subsequent occlusion of the injured vessel. In other cases the aneurysm increases, and by its pressure on the vessels may lead to gangrene of the limb. The same result may also be brought about by the giving way of the sac and the escape of its contained blood into the neighbouring parts.

ARTERIO-VENOUS ANEURYSM.

An arterio-venous aneurysm is an abnormal communication between an artery and a vein. There are two varieties. In the one, the communication between the two vessels is direct; in the other, the blood passes through an aneurysmal sac placed between the communicating trunks. The first variety is called an **aneurysmal varix**; the second a **varicose aneurysm.**

Arterio-venous aneurysms are usually the result of punctured wounds. They are generally situated on the limbs, and the communicating vessels are most frequently, in the case of the upper extremity, the brachial artery and the median basilic vein; in the lower extremity, the superficial femoral artery and its accompanying vein.

They are produced as follows:—A puncture is made by some sharp, narrow instrument, which passes through one vessel and opens the other. External hæmorrhage is arrested either by artificial means or else by the resistance offered by the surrounding parts. The blood flows in the direction of the least resistance, and this in the case of the artery is frequently into the wounded vein. In many instances the wounded vessels become adherent by the inflammatory effusion which is poured out for their repair, and the blood continues to pass directly from the one into the other—aneurysmal varix.

In other cases the extravasated blood separates the vessels from one another, and a traumatic aneurysm is formed between the artery and the vein; through this the blood circulates in its transit between the vessels—varicose aneurysm. Occasionally the arterial blood escapes not

only into the vein, but also into the surrounding tissues. The quantity effused may be sufficiently great to endanger the vitality of the limb.

More rarely, arterio-venous aneurysms are formed independently of injury, and sometimes result from the opening of an ordinary pathological aneurysm into a neighbouring vein.

The two forms of arterio-venous aneurysm are not equally serious. An aneurysmal varix often does not tend to increase in size, whilst a varicose aneurysm pursues the course of any untreated aneurysm, and tends to increase.

In either case, the veins below the communication become tortuous, distended, and thickened, whilst the arteries above it tend to become, not only dilated, but thinned to such an extent that their ligature, if attempted after the lapse of years, is extremely likely to be followed by secondary hæmorrhage. The distended veins pulsate almost as forcibly as do arteries, and the finger placed upon them at once detects a well-marked thrill. Over the seat of communication, a loud, continuous, humming bruit is always heard, which may be propagated for a considerable distance along the limb.

The chief danger of arterio-venous aneurysm is rupture and hæmorrhage. In some cases, independently of such an accident, the use of the limb is much interfered with.

ON THE CHANGES PRODUCED IN THE VESSELS BY THE VARIOUS METHODS EMPLOYED FOR THE ARREST OF HÆMORRHAGE.

All the methods of treatment employed for the arrest of hæmorrhage aim at one object, namely, its temporary arrest. By none of them can the vessel be permanently occluded; the most that ligature, torsion, acupressure, &c., can do is to take the place of the external clot, and arrest the hæmorrhage for a sufficient length of time to allow of the permanent closure of the injured artery.

Pressure acts mechanically, by obliterating the lumen of

the injured vessel, and so preventing the further escape of blood.

Acupressure is only another means of applying pressure.

Styptics induce the formation of an external clot.

Cold causes contraction of the cut artery and a narrowing of its lumen.

Ligature.—When a round ligature is applied to a vessel and tightly tied, the internal and middle coats, being

FIG. 44.

Artery soon after Ligature, showing the inner and middle coats retracted.

FIG. 45.

Artery filled with an Internal Clot.

FIG. 46.

Artery with Shrunken Internal Clot, and orifice sealed by fibrous tissue.

brittle, are cut across, and the external one, being tough and resistant, is drawn in by the ligature and obstructs the flow of blood. The *material* of which the ligature is composed exercises no influence on the behaviour of the vessel which is tied, but the *width* of the ligature is of importance. If it is round and narrow, the inner coats are certain to be cleanly cut. If broad and flat, the coats may not be cut at all, but may all be drawn together, as is the external coat alone when narrow ligatures are used. The permanent closure of the vessel by the exudation and organisation of plastic lymph, and the formation and organisation of an internal clot, does not differ from the process already described in dealing with the natural arrest

of hæmorrhage; and Ballance and Edmunds have shown that the mode of occlusion by fibrous tissue is the same whether the inner and middle coats have been divided by the ligature or not, although the amount of exudation and of fibrous tissue is less when these coats have not been divided than when they have been injured.

If the ligature be of soft animal tissue, such as catgut, and further, if it be aseptic, it will not act as a foreign body with respect to the artery which it encircles, and therefore suppuration will not ensue. If, however, the ligature be not rendered aseptic, then so long as it remains in contact with the vessel-wall inflammation will continue, suppuration will ensue, the artery-wall will ulcerate, and gradually the ligature will cut through the external coat and be cast off.

Torsion.—In torsion, the end of the cut artery is seized and twisted several times transversely to its long axis. The result of this treatment is the rupture of the internal and middle coats, as by the application of a ligature, and the twisting up of the external coat so as to form a sort of plug at the end of the torn vessel. Sometimes this twisted external coat sloughs, on account of the interference with its blood-supply, but this is not usually the case. The permanent closure of the vessel occurs in the manner already described.

RECURRENT AND SECONDARY HÆMORRHAGE.

Bleeding which re-commences after having been once arrested is called either recurrent or secondary. The term recurrent is sometimes limited to the hæmorrhage which occurs within twenty-four hours of injury, but the definition is purely arbitrary. Secondary hæmorrhage is most common during the first fortnight after injury, but may occur at any time before the wound is healed.

There are three main causes of secondary hæmorrhage, using the term to include the recurrent variety—first, **accident**; second, **unhealthy conditions of the wound**; third, **disease of the vessels**.

1. The accidental causes include such things as the use of a piece of bad ligature, which softens and gives way too soon; the overlooking of a wounded vessel, which does not bleed at the time the wound is inflicted, either because it contracts from exposure, or else because the patient is faint; the tying of a ligature badly, so that the knots slip; an accidental contusion of the wounded part, and the displacement or tearing open of recently formed clot or inflammatory exudation. If in any of these ways the ligatures which have been employed for the temporary arrest of hæmorrhage are displaced before the artery is permanently sealed, hæmorrhage will occur.

2. So long as a wound is healthy, and is either healing by first intention or by granulation, there is little fear of secondary hæmorrhage. The condition of the wound may be taken as the index of the conditions of the vessels and their contained clots. If the former is healing well, it is pretty certain that the ends of the arteries are being firmly plugged by fibrous tissue, and their internal clots are undergoing organisation. If, however, the wound is sloughy or the seat of erysipelas or other infective inflammation, then it is exceedingly probable that reparative processes are not progressing favourably in the cut vessel, and that the clot is tending to disintegrate. It is evident that under these circumstances secondary hæmorrhage is likely to ensue, for not only may there be a failure in the organisation of the inflammatory products, but the ends of the vessels themselves may be involved in the destruction, and may slough or become so much softened that the ligatures which encircle them may cut through. Further attempts at securing the artery may be thwarted by the fact that the softened vessels will not admit the application of a ligature, however carefully applied.

3. A diseased condition of the injured vessels predisposes to secondary hæmorrhage in two ways. First, when tied, the ligature is liable to quickly cut its way through the softened wall; or, when twisted, the twisted end is liable to slough. Secondly, on account of their unhealthy condition,

the reparative processes which ensue upon injury, and which normally seal a healthy vessel, are liable to be themselves of an unhealthy nature, and the inflammatory products either do not develop into fibrous tissue at all or do so only very slowly.

Injuries of veins.—Wounds of veins frequently heal without occlusion of the vessel, and, if clean cut, without any apparent cicatrix. A wounded vein heals more readily than a wounded artery, on account of the much more feeble blood-stream in it. The mode of healing is identical with that met with in the other soft tissues, namely, by plastic inflammation, with a consequent exudation and subsequent development of lymph into fibrous tissue.

AIR IN VEINS.

If air be mingled with the venous blood, it prevents the proper circulation of the latter through the lungs, obstructs the right side of the heart, and may cause death.

A wound in a vein does not permit the entrance of air except under certain conditions. If under ordinary circumstances a vein be incised, either the blood rapidly escapes from it or the atmospheric pressure causes its collapse. If, however, a vein be held open—*e.g.*, if in removing a tumour traction be employed at a time when a venous trunk is wounded, and especially if the wounded vein be near to the thorax, and its contained blood is brought under the influence of the suction produced by deep inspiration—then air is liable to pass with the blood-stream to the right side of the heart. The entrance of air is usually accompanied by a hissing or gurgling sound, and is immediately followed by urgent dyspnœa and laboured cardiac action. Such an accident most frequently occurs in operations at the root of the neck, where the veins are large, are close to thorax, and are more or less held open by the deep fascia.

CHAPTER XXIV.

FRACTURES.

A FRACTURE is a solution of continuity of a bone. If the broken ends of the bone are not exposed to the external air through the medium of a wound, the fracture is said to be a **simple** one. If the reverse is the case, the fracture is **compound.**

A **comminuted** fracture is one in which the bone is broken into more than two pieces.

A **complicated** fracture is one in which the fracture is complicated by some injury to important neighbouring structures—*e.g.*, the tearing of a main artery, extension into a neighbouring joint, dislocation of the fractured bone, &c.

Again, the direction in which the line of fracture traverses the bone supplies the means for further classification. Thus, a **transverse** fracture is one in which the bone is broken at right angles to its long axis; an **oblique** fracture, where the line of fracture is oblique. Simple transverse fractures are most common in children, and are usually accompanied by but little displacement.

UNION OF SIMPLE FRACTURES.

The fracture of a bone is followed by extravasation of blood, as in the case of injury to any other vascular structure, but here, as elsewhere, this effused blood takes no part in the healing of the injured tissue, and is itself completely absorbed in the manner already described in the chapter on Contusions.

The way in which a fractured bone is united differs in no essential particular from the healing of a flap wound by first intention, and, like the latter, is the result of a process of plastic inflammation set up by the injury. Soon after the fracture, the ends of the broken bone become hyperæmic, exudation ensues, new blood-vessels form, and the neighbouring bone is softened. The medullary canal, the space between the broken ends, the torn periosteum, and the surrounding soft tissues are thus filled with an interstitial granulation tissue of precisely the same nature as that which joins the flaps in the case of wounds of soft structures, but which in the case of bone is called **callus.**

In the chapter on Inflammation it has already been said that suppuration is caused either by persistent irritation or else by the presence of micro-organisms, and in simple fractures neither of these causes is usually found. Suppuration, therefore, is scarcely ever seen, and the inflammatory process set up by the injury stops short at the production of a sufficient amount of new material to unite the broken ends, and a sufficient softening of the osseous structure to ensure their proper welding together.

Of the inflammatory new formation, or callus, there are two varieties: the one is called "**definitive**" or **permanent**; the other, **provisional** or **temporary.** The definitive callus is that which lies between the fractured ends themselves; the provisional is found both within the medullary canal and outside the bone, beneath and around the periosteum.

If the callus be examined at various times during the union of fractures, it will be seen that it undergoes development into fibrous tissue just as does the granulation tissue in the healing of soft parts, but in the case of bone, on account of the influence exercised on all inflammatory products by the mother tissue, a further development into true bone occurs, in exactly the same way as in a wound of a tendon the inflammatory new formation, although at first only formed into fibrous tissue, is subsequently developed

into tendon. Occasionally in man, and commonly in animals, the developing callus passes through a cartilaginous stage.

The amount of provisional callus that is formed depends on the amount of movement to which the broken bone is subjected, and this is just what might be expected, considering that movement is likely to keep up the inflam-

FIG. 47.

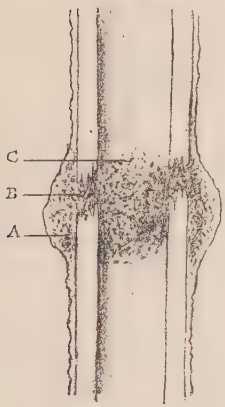


Diagram of a Fracture undergoing repair. A. External callus beneath the periosteum. B. Intermediate callus. C. Internal callus.

FIG. 48.

A Fractured Humerus three weeks after injury. The external and internal callus are ossified, but the intermediate callus has not yet been developed into bone.

matory process, and, as a consequence, to increase the amount of inflammatory exudation.

The callus which is first developed into bone is the provisional callus, and, in consequence, the broken ends become fixed by a mass of bone which encircles them, and by another mass within, which, passing right through the medullary canal of each fragment, greatly assists in keeping the ends in good apposition. Where, however, this

apposition is maintained by artificial means, such as splints, but little provisional callus is produced. After some weeks the definitive callus is formed, and from this time that which is temporary is gradually removed. Its total removal is necessarily the work of time, and the more of it there is the longer its removal will occupy. In consequence, it is sometimes many months, perhaps a year or more, before its obliteration is complete.

UNION OF COMPOUND FRACTURES.

A compound fracture may, and often does, unite in precisely the same way as a simple fracture. In other cases the process of union is complicated by suppuration.

Now, this is precisely what occurs in wounds of the soft tissues. Frequently the wound unites by first intention, *i.e.*, without suppuration; in other cases, suppuration and union by granulation result. The cause of the suppuration in the wounds of the soft tissues and of bones is identical. It is almost always the result of contamination by micro-organisms.

If, therefore, in the case of a compound fracture the wound be closed, and heal by first intention, the fracture will usually do the same, and a similar result may be obtained by dressing the wounded surface with antiseptics of various kinds, which prevent the influence of septic infection.

If, however, the wound be not closed, and become septic, then the inflammatory process will progress, an excess of effusion will result, pus will be formed, and the granulating ends of the bones will be exposed. Union will in time take place, at first in exactly the same way as in granulating wounds of the soft parts, namely, by the growing together of the two granulating surfaces, and subsequently by the formation of bone in the young fibrous tissue.

In some cases of compound fracture followed by suppuration portions of the bone die, or necrose. Such a result

is most frequent when the broken bone is comminuted and the fragments are detached from their vascular connections. When the bone dies, suppuration always continues, and union will be delayed until the necrosed bone has been separated and cast off. The subject of necrosis is dealt with at greater length in the chapter on Diseases of Bone.

NON-UNION OF FRACTURED BONES.

Occasionally, fractures fail to unite by bone, and, although the broken ends may be more or less fixed by fibrous tissue, such cases are spoken of as **ununited fractures.**

The **causes** of non-union are various, but may be grouped into two main classes—first, local; second, constitutional. Of these, the former are the more important, and will be first considered.

The **local** causes of non-union are three: (1) want of apposition; (2) want of rest; (3) want of blood-supply.

Want of apposition is probably the commonest cause of non-union. It may be brought about in various ways. In many cases the contraction of muscles inserted into the fractured bone separates the fragments, as is well seen in fractures of the coracoid or olecranon processes, of the condyles of the humerus, of the neck of the femur, &c. In other instances portions of muscle may be caught between and keep apart the broken ends. In the case of transverse fracture of the patella, more causes than one are in action, for the broken ends are separated both by the contraction of the quadriceps extensor, and to a great extent by the effusion of fluid into the knee-joint, which floats the fragments up and keeps them apart.

Want of rest is of less importance than want of apposition, but it occasionally prevents bony union. If callus has been formed and is in process of organisation, there can be no doubt that the recently formed fibrous and osseous tissue may be readily broken down by careless movement. It will, however, be again reproduced, and it is only when this

breaking down has frequently occurred that bony union fails; for, in the large majority of cases in which a fracture remains ununited from want of rest, firm union will rapidly result from adequate surgical treatment.

Want of blood-supply is very seldom the sole cause of non-union. In fractures of the lower third of the humerus, the comparative frequency of non-union has been attributed to the fact that the line of fracture traverses the nutrient artery; and the failure of union of intra-capsular fractures of the femur is also attributed to the scanty blood-supply to the head of the bone. It is more probable that the fracture of the humerus fails to unite from want of rest, and that of the neck of the femur from failure of apposition.

Interference with the circulation by *venous thrombosis* in the neighbourhood of the fractured bone is believed by many surgeons to play an important part in the failure of union of fractures. It is certainly true that, in cases of non-union, there is very frequently a persistent œdema of the injured extremity, and it is possible, though difficult to prove, that the thrombosis which causes the œdema may also prevent the union of the fractured bone.

Constitutional causes of ununited fractures.—Of all the constitutional diseases which tend to prevent union of fractures, **scurvy** is probably by far the most active, for it would appear, from the descriptions of authors who have had opportunities of watching cases of scurvy, that, so long as the patient is suffering from this disease, fractures are very likely to remain ununited. Next to scurvy, **chronic nephritis,** in any of its various forms, appears to delay union more effectually than disease of the other viscera; and lastly, it may be said that any disease which induces debility, and which perhaps, in addition, causes an alteration in the naturally healthy bones themselves, will tend to delay, if not to prevent, union. Causes such as these, however, act in a manner which is very easy of comprehension. If the patient be in an enfeebled condition, he is not likely to

repair any injury with rapidity, and, in consequence of his lowered vitality, the inflammatory process does not result in the production of a sufficient amount of callus, or a sufficient softening and welding together of the fractured bones.

Changes in the bones in ununited fractures.—If a fractured bone fails to unite, the broken ends may be either merely held in apposition by fibrous tissue, or a false joint may be formed.

FIG. 49.

Ball-and-socket Joint formed after Intra-capsular Fracture of the Neck of the Femur.

The inflammatory effusion, which has been poured out after the injury, undergoes organisation into bone, in some parts at least, and, as a result, the medullary canal is occluded, and the fractured ends become rounded off and smooth. Where the fragments are widely separated, there may either be no uniting material at all, or else it may be so thinned and stretched as to deserve the name of "**membranous union.**" Where the uniting medium is stronger and denser, the union is said to be "**fibrous**" or "**ligamentous.**"

In some instances, when the ends of the fractured bone have been placed in apposition, and move on one another,

a **false joint**, or **pseudarthrosis**, results. In such cases the fragments are enclosed in a kind of capsule of fibrous tissue, the inner surface of which becomes, in time, smooth and polished, and secretes a serous fluid which facilitates movement. The ends of the bones become variously altered in shape and adapted to one another, but the false joint is either a simple hinge or else a ball-and-socket joint. The latter condition is well shown in the accompanying drawing, from a case of ununited fracture of the femur.

When a false joint has been formed, or when the fractured ends have become rounded, smooth, and hard, union by bone will never occur, however long the bones are kept in apposition and at rest. The opportunity for union has passed by, and the chance has been missed. When the bones were softened by recent inflammation and embedded in recent granulation tissue, then was the time for union to take place, but if, after the inflammatory products have become organised and the bones hardened, union has not resulted, it can never take place, unless by artificial means fresh inflammation is excited.

SPONTANEOUS FRACTURES.

When a bone breaks from the application of an amount of violence which would not break a healthy bone, the fracture is said to be "spontaneous." The violence may be extremely slight, and almost unnoticed by the patient. A diseased state of the osseous structure predisposes to such fractures. The bone may be simply in an advanced state of senile decay and degeneration, or, on the other hand, and much more frequently, may be the seat of a malignant tumour. Such a tumour, if primary, is sarcomatous, but if secondary, it may be carcinomatous. Thus, it is not uncommon to meet with cases of spontaneous fracture in patients suffering from cancer of the breast, and very often there is no history of pain or swelling in the broken bone antecedent to its spontaneous fracture. In addition to these causes, it

has recently been pointed out that in tabes dorsalis the bones are sometimes very brittle, and may break spontaneously. Both mollities ossium and rickets may cause the bones to become so much softened that they break on the application of the slightest violence, and, more rarely, the bones of a limb which is paralysed or has been kept at rest for very long become softened to a similar extent. In spontaneous fractures, failure of union by bone is common, though an exception must be made for the tabetic cases, in which union is generally firm.

SEPARATION OF EPIPHYSES.

The separation of an epiphysis is an accident which necessarily occurs only in children and young subjects. In this form of injury, both the epiphysis and the epiphysial cartilage are separated from the diaphysis, the epiphysial cartilage itself being seldom torn. The injury is repaired in the same way as is a fracture, and it is exceptional to find that, as a result of the separation of the epiphysis, the subsequent growth of the bone is impeded. Sometimes, however, growth is partially arrested, and the affected limb may thus become shorter than its fellow.

Both congenital syphilis and scurvy rickets predispose to separation of the epiphyses.

CHAPTER XXV.

DISLOCATIONS.

The term dislocation simply implies a displacement, but, used without any qualifying adjective, is taken to mean the displacement of one articular surface from another with which it is naturally in contact.

Dislocations are commonly the result of violence applied to a limb at a distance from the joint which is the seat of the displacement. Thus, whilst a fall on the shoulder or hip may cause a fracture, dislocation of these joints is usually produced by violence applied to the arm or leg. Dislocations are also caused by irregular muscular action; *e.g.*, the lower jaw is frequently displaced by a sudden contraction of the muscles of mastication at the time when the mouth is opened, as in the act of yawning or laughing. The patients in whom dislocations are most frequently met with are young and muscular adults, for in old people the bones yield rather than the ligaments of the articulation, and in children the joints are so supple that dislocations are not readily produced.

It is evident that in the production of a dislocation some of the soft structures around the damaged joint must be injured, and thus we find that the capsular and other ligaments are torn, together with the surrounding muscles and tendons, to a varying extent.

CHANGES IN AN UNREDUCED DISLOCATION.

If the dislocation be not reduced, certain changes occur in the articular bone and the surrounding soft tissues which are of considerable practical importance.

The immediate results of the injury are a synovitis of the damaged joint and an effusion of blood into it and the tissues around; both of these rapidly subside. The torn ligaments, muscles, and tendons soon undergo repair by fibrous tissue, and become matted together by the formation of "adhesions" in such a way that various important structures, such as vessels and nerves, become adherent to the muscles and tendons amongst which they lie, and are dragged upon by any force which exercises traction on the latter. In consequence of this, if violent efforts are made to reduce the displaced bone after the newly formed adhesions have become firm and strong, either the vessels or nerves to which they have formed attachments may be ruptured.

The cartilage of the dislocated bone undergoes degenerative changes which result in its metamorphosis into fibrous tissue, and, if it rests on another bone, further changes may ensue which terminate in the production of a false joint. Take, for instance, the case of a subcoracoid dislocation of the humerus. The head of the latter bone will gradually alter in shape, and become flattened, and the neck of the scapula on which it rests will be hollowed out. In each bone the alteration in shape is the result of pressure, for in all tissues continuous pressure produces atrophy. Around the newly formed depression in the scapula a rim of new bone is then formed, for the irritation produced by the presence of the displaced humerus causes a formative periostitis in the neighbouring bone. The same irritation causes the condensation of the neighbouring soft structures, and by this means a fibrous capsule is formed, and the false joint is thus completed. Meantime, changes have been in progress in the glenoid cavity, its cartilage has become fibrillated, and from its floor a new growth of fibrous tissue springs which gradually completely fills the cavity itself.

At first the patient has but little use in a limb one of the chief joints of which is the seat of an unreduced dislocation.

Gradually, however, it becomes less painful and stiff, and after many months the part is frequently restored to much of its former power, for the muscles accommodate themselves to the altered relations of the neighbouring structures, and as the new joint is formed, and the inflammation set up by the injury subsides, pain on movement slowly passes away.

A consideration of the changes just described will show at once that, apart from the dangers of reduction of old dislocations, there comes a time when replacement of the dislocated bone is no longer possible, on account of the fixation of the bone in its new position, and the filling up of the articular cavity which it formerly occupied.

CHAPTER XXVI.

REPAIR OF MUSCLE, TENDON, CARTILAGE, AND NERVE.

REPAIR OF MUSCLE.

Muscle is a tissue which in man is practically never reproduced, and in animals only to a very slight extent, the amount of new muscle fibres being in any case quite microscopic. Consequently, in wounds of muscle the divided portions are never united by newly formed muscle, but always by fibrous tissue, which is produced in the manner already described in dealing with union of wounds. The separation of the divided muscle during the healing process naturally influences the amount of scar tissue formed, and the greater the separation the more powerless is the muscle after cicatrisation has been completed. It is therefore of importance to maintain as good apposition as possible during the healing of the wound.

REPAIR OF TENDON.

A divided tendon placed under favourable conditions unites by tendon in the following manner: —Plastic exudation takes place into the tendon-sheath and into the cut ends of the tendon themselves, the exudation being vascularised, and subsequently developed into fibrous tissue, in exactly the same way as is the granulation tissue of a flap-wound. After this a further development into tendon tissue occurs, and the previously irregular masses of fibrous tissue are gradually shaped into longitudinal bundles, with connec-

tive-tissue cells placed between. Finally, the tendon, which is at first adherent to its sheath, is gradually separated, and again becomes free and movable.

If the ends of the divided tendon be allowed to retract to considerable distances, or if much suppuration occurs, the tendon will not unite as above described. On the contrary, its ends will become adherent to the surrounding parts, and the use of the muscle which is inserted into it will be lost.

REPAIR OF CARTILAGE.

Cartilage, like muscle, is not reproduced in cases of injury. The breach is repaired by fibrous tissue.

INJURIES OF NERVES.

If a nerve is divided, certain changes occur in both its peripheral and proximal extremities, and also in the structures to which it is distributed.

The proximal end of the divided nerve becomes swollen and bulbous, the enlargement continuing to increase for several weeks after section. The bulb is oval in shape, about two or three times as thick as the divided nerve-trunk, and about twice as long as it is broad. It is tougher than normal nerve tissue, and adherent to the structures amongst which it is placed. On section, it presents a fibrous appearance, but bundles of nerve tubules can be seen embedded in its substance. On microscopical examination, it is found to consist of numerous young nerve-fibres lying in a matrix of fibrous tissue.

The peripheral end of the divided nerve undergoes atrophic and degenerative changes at the same time that the bulb is being formed. The myelin breaks up, collects in globules in the nerve-sheath, and is finally entirely removed. The axis-cylinders disintegrate, and in the course of a few weeks at the latest are completely destroyed, As a result of this destruction, the peripheral end of the nerve is very perceptibly diminished in size.

The manner in which a severed nerve unites is variously described by different authors. It is probable that the following description is correct.

After undergoing the degeneration just described, the peripheral end regenerates. The axis-cyclinders are first formed from the proliferated nuclei of the sheath of Schwann; around these a myelin sheath is subsequently developed, and, if the peripheral end is in contact with the proximal extremity, the young nerve fibres formed in the bulb of the latter unite with the newly formed axis-cyclinders in the lower end, and nerve functions are restored. It must, however, be borne in mind that union will not occur unless the divided extremities are maintained in close contact, and as nerves, like all other soft structures, are in a state of longitudinal tension, it is usually necessary to suture the cut ends in order to maintain the necessary apposition.

The structures to which a nerve is distributed suffer in various ways if the latter is divided.

The skin, in addition to losing its sense of touch, becomes glossy and shiny, ulcers may appear at the peripheral parts, and occasionally small portions of tissue slough. In the case of the hand, painless whitlows may form, and the nails may either be cast off or become fibrous and brittle, with exaggerated curvatures and transverse ridges and furrows. Immediately after section, and for a time which may extend to two or three weeks, the anæsthetic parts are hyper-vascular and warmer than natural, on account of the division of the vaso-motor fibres. Very soon, however, this condition gives way to an abnormal coldness, and the loss of heat frequently amounts to five or ten degrees.

The joints supplied by the severed nerve are sometimes swollen and painful shortly after an injury; later on, they are often partly anchylosed.

The muscles are paralysed from the moment that the nerve is divided, and undergo an extremely rapid degeneration and atrophy. Faradic contractility is lost within a

few days, and galvanic irritability in from three to six weeks. The muscle substance itself is the seat of fatty degeneration, and within two or three months of the injury the entire muscle is usually completely atrophied.

Notwithstanding the extent to which these so-called "trophic" changes may proceed, a complete restoration of function will result if the divided nerve becomes united at a subsequent period, and not only will sensation be restored, but atrophied and paralysed muscles will be renovated.

Division of a nerve is not always followed by degeneration of the peripheral end before union takes place, and, in animals at any rate, a "union by first intention" without antecedent degeneration has been described. Clinical experience tends to show that, although such union may, and does occasionally, occur in man, it is rarely seen, and, from the very nature of the process, opportunities for examining the recently joined nerve are very rarely forthcoming. A year or two sometimes elapses before sensation and motion are restored.

CHAPTER XXVII.

DISEASES OF ARTERIES. DEGENERATION AND ATHEROMA.

PRIMARY FATTY DEGENERATION.

Fatty degeneration may be either primary, or secondary to pre-existing inflammation. Primary fatty degeneration may affect any of the coats of a vessel, but is most common in the inner coat. Here it first attacks the endothelial lining, and the cells of the latter are gradually filled with granules of fatty matter; the diseased cells disintegrate and are cast off. The degenerative process sometimes extends over considerable areas, and is most commonly met with in the aorta. It may be distinguished from atheroma by observing that the degeneration is limited to the endothelial surface, and that the deeper parts of the inner coat are healthy. When fatty degeneration affects the deeper parts of the inner coat or the middle and external coats, the whole artery is liable to become greatly weakened, and where not supported adequately by the surrounding tissues, as in the brain, the diseased condition may lead to rupture and hæmorrhage; degeneration of the middle and outer coats is most common in the smaller arteries and arterioles. Fatty degeneration in all its forms is essentially a disease of late life.

PRIMARY CALCAREOUS DEGENERATION.

Calcareous degeneration of an artery may either be secondary to pre-existing inflammation, or may occur primarily in vessels otherwise healthy. The subjects of

primary calcareous degeneration are always past middle age; the vessels most frequently attacked are those of medium size, especially the tibials and the arteries at the base of the brain. The degeneration affects more particularly the middle coat, and the individual muscle fibres of the latter are gradually infiltrated with calcareous salts. The calcifying process, following the circular arrangement of the muscle fibres, causes the production of rings of calcareous material which are very typical of the primary form of calcareous degeneration. After a time both the inner and middle coats may become similarly diseased, and the whole vessel may be converted into a rigid and brittle tube. The circulation through a calcareous vessel is necessarily greatly impeded, and, occasionally, thrombosis may occur from the clotting of blood on the roughened inner coat.

FIG. 50.

Primary Calcareous Degeneration of the Femoral Artery, showing the rings of calcareous material which are formed in the muscular coat.

Fatty and calcareous degenerations frequently co-exist in the same artery.

ACUTE ARTERITIS.

Acute inflammation of arteries never occurs idiopathically, and, when it has begun, shows no tendency to spread. If a vessel be injured, its coats will inflame, exudation will follow, and the artery will be occluded in the way already described in the chapter on Hæmorrhage. The most typical examples of acute arteritis, however, are supplied by vessels lying in the midst of suppurating tissues. It is true that arteries offer very considerable resistance to the spread of

inflammation, but, nevertheless, they are frequently involved in the process. The blood in the inflamed vessel clots, the artery-wall is softened by exudation, suppuration follows, and all the coats are gradually ulcerated through. In most cases no hæmorrhage ensues, for the vessel is already full of clot, and above the seat of suppuration it is thickened by plastic exudation. In some cases, however, and more particularly when the inflammatory process is an infective one, large portions of the artery slough, and the clot being soft and disintegrating, and insufficient to arrest the flow of blood, copious hæmorrhage may result.

The lodgment of septic emboli in vessels is another cause of acute inflammation. In cases of ulcerative endocarditis, portions of fibrin infiltrated with pyogenic micro-organisms are liable to be detached and swept from the aortic valves into the blood stream. Wherever they lodge they excite a destructive inflammation similar to that which is in progress at the place from which they have been derived, and, in consequence, the wall of the vessel is liable to become either merely softened by inflammation, and subsequently dilated, so as to form an aneurysm, or else penetrated by ulceration. In some cases hæmorrhage results, but in many others the occlusion of the artery by clot prevents the escape of blood.

Acute inflammation of the aorta has been described by various authors. This form of inflammation, which is supposed occasionally to follow typhus fever, does not result in suppuration. There is said to be a gelatinous exudation into the inner coat, the endothelial surface of which may be roughened by deposit of fibrin. It is certainly of rare occurrence.

A spreading inflammation of arteries resulting in their occlusion has also been described under the name of "obliterative arteritis." There is, however, no good evidence that arteries are thus affected in the absence of syphilis, and in the more typical cases described under this name an exostosis was present which compressed the sub-

clavian artery and the brachial plexus, and probably caused the interference with the pulse which gave rise to the belief that the arteries were occluded.

ATHEROMA, OR CHRONIC ARTERITIS.

Atheroma is not a degenerative, but an inflammatory process. A better name for it is chronic arteritis.

Speaking generally, the chief **cause** of chronic arteritis is **increased blood-pressure**. This may be brought about in various ways. Laborious occupations, and more particularly those which call for sudden and long-continued strain, undoubtedly tend to cause atheroma, and sudden strains are even more likely to give rise to trouble in those who usually lead a quiet and sedentary life, and who for short periods only, and at long intervals, follow pursuits which call for unusual exertions.

It is well known that in chronic interstitial nephritis the whole blood-pressure is considerably higher than natural, and thus patients who suffer from granular kidneys are rendered more liable than others to chronic arteritis. Again, in those forms of heart disease which lead to hypertrophy, the increased power of the heart may cause a greatly increased strain on the aorta, and thus produce atheroma in that vessel. Atheroma is more common in drunkards than in sober individuals. This may be accounted for in part by the diseases of the kidneys and liver which are induced by intemperate habits, and may also result from the direct influence of alcohol on the blood-vessels themselves. The arteritis which is caused by syphilis is described at p. 242.

Considering that increased blood-pressure is the chief cause of atheroma, we should naturally expect the latter to be most common in the larger arteries, where the blood-pressure is greatest; and such is the case. The aorta is diseased more frequently than is any other vessel, and the arteries of the limbs may be said to be affected almost in proportion to their size. Atheroma may be met with in

people of but twenty or thirty years of age, but is most common after middle age.

The changes that take place in an atheromatous artery are such as might be expected, considering that the process is an inflammatory one. Examined at its earliest stage, the disease is seen to cause slight swelling of the internal coat, which bulges toward the lumen of the vessel. Microscopical examination will show that exudation of inflammatory products has occurred, and the cellular tissue beneath the endothelium is seen to be the seat of a considerable collection of round cells. These have collected here, partly because the inner coat is the first to feel the effects of the undue tension, and partly also because on account of the looseness of the sub-endothelial cellular tissue, there is more room for the inflammatory exudation to collect in this situation than in the middle and external coats. It is, however, quite erroneous to suppose that the latter tunics escape entirely. Such is not the case. On the contrary, cell exudation takes place into them also, and increases and extends as the atheromatous process progresses, so that the inflammation may extend beyond the outer coat and may involve the sheath.

It is probable that in some cases the inflammatory process undergoes resolution and the exuded cells are removed. Frequently, however, this does not occur, aud the cells either undergo degenerative changes or develop into fibrous tissue.

Very frequently, indeed, the degenerative process predominates in the cell exudation in the inner coat, and for the reason that in this situation the cells do not receive a sufficient blood-supply. It must be remembered that the vasa vasorum do not pass beyond the middle coat, that the inner coat is mainly nourished by a process of osmosis, and that such a process is only capable of nourishing a certain limited thickness of tissue. When, therefore, the blood-vessels which have been formed during the first stage of the inflammatory process shrink and disappear, the

cell-growth which they have produced is left without sufficient means of nutrition, and consequently degenerates. In most cases the degeneration is of a **fatty** nature, and the cell collection is gradually converted into a yellow caseous mass. The endothelium also undergoes degenerative changes which result in the destruction of its constituent cells; these, after a time, peel off and lay bare the subjacent diseased coat. In consequence of this, the disintegrating caseous mass is exposed to the blood-stream, and by it is slowly washed away, the wall of the vessel being thus left roughened and partly destroyed.

In the meantime, the cells which have collected in that part of the inner coat which is in contact with the muscular tunic, as well as those which lie in the middle and outer coats, have undergone organisation into fibrous tissue, and by this means the arterial wall is strengthened at the weak spot, the coats are matted to one another, and the artery in time becomes adherent to its sheath. For this reason, although the blood obtains an entrance into the coats, it does not separate them from one another, and form a dissecting aureurysm; neither, as a rule, is its force sufficient to rupture the diseased vessel. But although fatty degeneration is the most common sequel of the cell exudation, it does not always occur, and even when present is frequently associated with other changes. Very frequently **calcareous** degeneration of the inflammatory products either takes the place of, or succeeds to, the caseation, and, in consequence, irregular plates of calcareous matter develop in the arterial wall. They present an altogether different appearance to the rings caused by primary calcareous degeneration of the muscle-coat, already described.

But, whilst degenerative processes are very common in the cells exuded into the inner coat, it must not be thought that development of fibrous tissue never results. It is probably much more common than is generally supposed, and very frequently, indeed, some attempt at the formation of fibrous tissue may be found occurring together with

caseation, whilst, in some instances at least, the whole cell mass undergoes development, and the arterial wall is permanently thickened and indurated. The greater the tendency to the formation of fibrous tissue, the less likely is the endothelium to perish.

To the naked eye, an artery which is atheromatous is at once seen to present a very irregular and lumpy inner surface, to be more rigid than natural, and, when cut across, to remain distended, and not to contract like a healthy vessel. Closer inspection will show that the endothelium is raised in numerous patches, and, through the lining membrane of cells, the subjacent material looks yellow or opalescent. The yellowish substance is caseous material lying beneath the endothelium, and is readily exposed by peeling away the latter. If fibrous tissue has been formed, the yellow tint is less marked, and the endothelium is more adherent.

FIG. 51.

An Atheromatous Artery. In the lower part of the specimen, the intima is irregular and rough. In the upper part, the intima has been destroyed, and a bulging of the other coats indicates the commencement of a sacculated aneurysm.

At a later stage, the artery is found to be distinctly roughened, the endothelium has perished, and the deeper parts of the intima, or even the muscle-coat, may be exposed to the blood-stream. Shreds of fibrin may adhere to the roughened spots, or definite coagula may have formed. When calcification has occurred, the artery is more than usually rigid, and irregular plates of calcareous matter can be both seen and felt in its inner coat. The sheath is more adherent than natural, and when rolled between the fingers the vessel feels firm and cord-like.

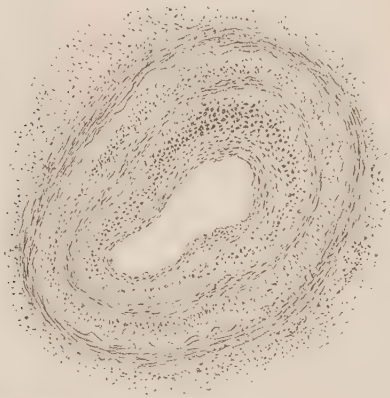
In consequence of the alteration in its structure, the

circulation through an atheromatous vessel is more or less impaired. The diseased artery is more rigid than natural, and, having lost its elasticity, it is unable to accommodate itself to the constantly altering requirements of the tissues for blood, and the parts to which its branches are distributed become ill-nourished, atrophied, or degenerated. Worse results than this may ensue, for the roughening of the inner coat may cause deposition of the fibrin from the flowing blood, and occlusion of the vessel by thrombosis may completely arrest the circulation.

SYPHILITIC ARTERITIS.

Syphilitic arteritis is characterised by the great thickening of the inner coat. It may occur at any time after inocula-

FIG. 52.



Syphilitic Disease of one of the Cerebral Arteries. The inner coat is thickened by fibrous tissue, and the lumen is much narrowed. (Crouch: $\frac{1}{2}$ inch.)

tion, and, though most common in the cerebral vessels, is not confined to them, but affects the small arteries of any part which is the seat of syphilitic inflammation, and is especially common in the neighbourhood of gummata.

Microscopical examination shows that there is a growth

of granulation tissue in the inner coat, with the subsequent formation of fibrous tissue to such an extent that the intima may become three or four times as thick as the middle and outer coats. These latter also are the seat of some cell exudation, but do not become so greatly thickened. As the result of this formation of fibrous tissue, the lumen of the vessel is sometimes much narrowed, and in other cases the endothelium is so altered by the exudation beneath it that the blood clots on it, and the artery becomes occluded by a thrombus. Such interference with the cerebral circulation is liable to cause "softening of the brain." Some authors consider that similar arterial disease accounts for the degeneration and caseation of gummata. When syphilis attacks the larger vessels, it not infrequently leads to the formation of aneurysm.

CHAPTER XXVIII.

DISEASES OF ARTERIES.

ANEURYSM.

An aneurysm is "a tumour containing blood, and communicating with the cavity of an artery."

The **causes** of aneurysm are practically those of atheroma, for pathological aneurysms, with which alone we are at present concerned, are almost invariably preceded by disease of the vessel. Therefore, laborious occupations, disease of the kidneys, and syphilis are amongst the most common of the accepted causes of aneurysms. The formation of traumatic aneurysms has already been described.

It is very easy to see how chronic arteritis may cause aneurysm, for the inflammatory process frequently weakens the vessel, and so favours its distension by the blood. In other cases it is the loss of elasticity rather than the weakening which brings about the aneurysmal dilatation, for, if the heart's action be sufficiently strong to dilate the rigid vessel, toughened by fibrous tissue, the artery will, in consequence of its loss of elasticity, remain dilated, and thus form an aneurysm.

In the presence of one of the above causes, the locality of an aneurysm is determined by local conditions. The aorta is more frequently implicated than other vessels, on account of the greater blood-pressure within it, whilst the slight arrest of the blood-stream at the point of bifurcation of any vessel tends to the formation of an aneurysm above such a point. Again, vessels at the flexures of limbs are

more liable to become aneurysmal than vessels in other situations, and for the reason that, in any artery which is diseased, sudden and forcible bending may cause injury to the brittle internal and middle coats.

The aneurysms that occur in children and young adults are most probably the result of the **lodgment of emboli**. The exact manner in which emboli cause aneurysms is yet a matter of dispute. According to one theory, the artery becomes dilated above the seat of embolism through the increase in the blood-pressure above the seat of plugging. This is most probably incorrect, for, were such an explanation true, aneurysms would develop with comparative frequency after ligature, whereas although they may follow occlusion of the vessel by surgical means, they do so with extreme rarity, and are more frequently the result of a failure of the proper occlusion of the artery than of its obliteration. According to another theory, an embolus may cause aneurysm by setting up inflammatory changes in the vessel at the point at which it lodges. The inflammation may result either in the complete penetration of the artery by a process of ulceration, or else in softening of its coats and subsequent dilatation. All emboli do not act in this manner, and the production of an aneurysm in any given case is attributed either to the embolus being calcareous and rough, or else to its being derived from a heart affected with "ulcerative endocarditis," and consequently itself impregnated with the products of an *infective* inflammation.

Lastly, another possible cause of aneurysm must be mentioned. Suppuration around a vessel may so damage it that the coats may yield and an aneurysm form, or the artery may give way and rupture.

VARIETIES OF ANEURYSM.

An aneurysm consists of a **sac** and its **contents**. The sac is formed by the coats of the diseased vessel, its sheath, and the surrounding structures, in varying proportions.

The contents of the sac are fluid blood, clotted blood, and laminated fibrin.

Pathological aneurysms have been classified in various ways. The following appears to be one of the simplest:—

(1) Fusiform aneurysm.
(2) Sacculated aneurysm—
    (*a*) True;
    (*b*) False;
    (*c*) Diffused.
(3) Dissecting aneurysm.
(4) Cirsoid aneurysm.

The various forms of traumatic aneurysm have been already described in the chapter on Injuries of Vessels.

A fusiform aneurysm, or an aneurysmal dilatation, is a dilatation of a vessel in its whole circumference and for a considerable portion of its length. Such an aneurysm is oval in shape, and continuous at each end with the cavity of the vessel on which it has been formed. Its sac consists of the arterial coats in an atheromatous state, with a few shreds of adherent fibrin. It contains usually little else than fluid blood. Fusiform aneurysms are found on the large arteries alone, and far more frequently on the aorta than on any other vessel. (See Fig. 53.)

A sacculated aneurysm is a dilatation of an artery in a part only of its circumference. A "**true**" sacculated aneurysm is one the sac of which consists of all three coats of the vessel. A "**false**" sacculated aneurysm is one in which the internal and middle coats have been thinned away, so that, on section, the sac is found to contain the outer coat alone. (See Fig. 54.)

Considering that the chief cause of aneurysm is atheroma, it is evident that "true" aneurysms must be of rare occurrence, for the atheromatous process itself usually causes destruction of some of the coats before an aneurysm commences.

A diffused sacculated aneurysm is one in which the

sac contains none of the coats of the vessel, but consists simply of the sheath, with condensed surrounding tissues and blood-clot. This form of aneurysm has also been called "consecutive."

FIG. 53.

A Fusiform Aneurysm of the Common Femoral Artery.

FIG. 54.

A Sacculated Aneurysm of the Popliteal Artery.

A dissecting aneurysm is one in which the blood is contained in a sac formed within the wall of the artery itself, the blood generally passing down between the layers of the middle coat, and splitting the wall of the vessel for a greater or less distance. Such an aneurysm necessarily results from a destruction of the inner coat, for by this means alone can the blood obtain an entrance. The reason why dissecting aneurysms are not of greater frequency is that, as a rule, the coats are matted to one another by fibrous tissue before the inner coat gives way. It is when this process of adhesion fails that a dissecting aneurysm is formed. Dissecting aneurysms occur in the aorta alone, and may progress in one of two ways. In the first place,

the blood may burst its way through the intima, and may thus return to the cavity of the diseased vessel after a transit of some length through its walls. Secondly, it may burst through the outer coat, and, escaping into the surrounding parts, cause death by hæmorrhage.

The natural course of an aneurysm may be in one of two directions. It may progress, and cause the death of the patient, or may become spontaneously cured. Unfortunately, the former is infinitely the more common, yet it is of much importance to thoroughly understand the processes by which, independently of surgical interference, a cure may be promoted.

A fusiform aneurysm sometimes remains in an almost stationary condition for many years, and so long as it remains fusiform is little likely to kill the patient, unless, indeed, it do so by interference with the action of the heart. What usually happens is that, after a varying length of time, the dilated artery commences to yield more rapidly at one part of its circumference than at the rest, and thus from the fusiform aneurysm a sacculated aneurysm springs.

The sac of a sacculated aneurysm at first consists of some at least of the coats of the diseased vessel, although even from its commencement it receives support from its sheath and from the surrounding structures. As the sac increases in size from the constant pressure of the blood, the arterial walls become more and more thinned away, so that, although at first perhaps a true aneurysm, its inner and middle coats wear away and it becomes a false one. Later still, all its coats being destroyed, it is called "diffused" or "consecutive," the blood being limited merely by the arterial sheath and the surrounding tissues. These latter undergo considerable changes, and become matted together by fibrous tissue into a firm, resistant mass. Were it not for the support thus accorded to the sac by the surrounding soft tissues and by the arterial sheath, the rupture of an aneurysm would occur at a much earlier period than is usually the case. This point is well illus-

trated by aneurysms of the cerebral vessels, for the latter, receiving but little or no support from the tissues in which they lie, are never the seat of aneurysms of any size; the diseased vessel gives way instead of continuing to dilate.

In any case, when an aneurysmal sac no longer contains any of the arterial coats, and consists merely of the condensed surrounding tissues, it is very liable to increase with greater rapidity than before, and, inasmuch as the support it receives from its surroundings necessarily varies at different parts, it increases more rapidly in the direction of the least resistance. After a variable time it bursts, and is now called a **ruptured aneurysm**. The rupture may take place into the soft tissues of the part in which the vessel is placed, and, if the loss of blood be great, the rupture may be followed by such an amount of obstruction to the circulation that gangrene results. Sometimes the rupture takes place into a serous cavity, such as that of the pleura or pericardium, and then the escape of blood is at once both rapid and profuse. Sometimes, though comparatively rarely, an aneurysm reaches the cutaneous surface, and bursts there. If so, the hæmorrhage is not so sudden or profuse as would naturally be expected. On the contrary, days or weeks may pass before death results. During its growth an aneurysm spares no structure with which it comes in contact. The constant pressure of the sac, by interfering with their circulation, causes atrophy of soft tissues and of bones alike, and thus, in the case of aortic aneurysms, the bodies of the vertebræ, the ribs, and the sternum may be in great part destroyed. In popliteal aneurysms, the posterior surface of the femur is sometimes partially excavated, and the sac has been known to open into the knee-joint.

The pressure exercised by the aneurysm on the surrounding structures occasionally causes tolerably acute inflammation. This may progress to suppuration and sloughing, the sac may be opened, and profuse hæmorrhage may cause a speedy death.

SPONTANEOUS CURE OF ANEURYSM.

There are practically but two ways in which an aneurysm can become cured; the one is by the gradual filling of the sac with laminated fibrin, the other by the rapid clotting of the blood contained within the sac. A clot composed of laminated fibrin is often called an "**active clot,**" whilst that formed by the coagulation of the blood contained in the sac at any one time is called a "**passive clot.**" In either case, the sac having become filled, the clot extends into and occludes the diseased vessel from which the aneurysm springs. All treatment, medical and surgical alike, aims at causing the formation of either an active or a passive clot.

For the formation of a laminated clot it is essential that the blood shall continue to circulate through the sac, and that its flow shall not be sufficiently forcible to wash away the fibrin.

The more feeble the stream of blood, the less is it able to dilate the sac; and the more fibrin is deposited on the walls of the latter, the more capable will they be of resisting the blood-pressure. Now, the presence of an aneurysm on a vessel more or less impedes the flow of blood through it, and, consequently, the peripheral parts tend to become badly supplied. But in all parts of the body the obstruction to the circulation through any one vessel causes the blood to betake itself to other and less obstructed channels, and a "collateral circulation" is established. This also occurs in the case of aneurysm, and, as the blood flows through other vessels, the circulation through the diseased artery becomes more feeble, and the tendency to the formation of a laminated clot is increased. The more feeble the patient becomes, the less is the blood-tension and the weaker the circulation, and so, in some cases at any rate, a natural cure takes place.

In addition, however, to this process of spontaneous cure —a tendency to which may be said to exist in all aneurysms —certain accidental circumstances may arise which favour

the occlusion of the aneurysmal sac. The first of these accidents is the separation of a portion of the clot already formed in the sac, followed by its lodgment either in the mouth of the sac itself or else in the vessel on the distal side of the aneurysm. In the latter case, the artery being occluded and the circulation through it arrested, the collateral circulation will be opened up, less blood than formerly will flow through the aneurysm, and the latter may then be filled with laminated clot. It is evident that for such a curative process it is essential that the arteries which carry on the collateral circulation shall be given off on the cardiac side of the aneurysm, for if such is not the case, and the collateral branches come off between the mouth of the sac and the place where the vessel is plugged, just as much blood will pass through the diseased artery to reach the enlarged collaterals as previously passed along the main trunk itself.

This method of natural cure by occlusion of the artery beyond the sac is imitated by the surgeon in the operation of distal ligature, an operation which only holds out hope of success when no important vessels are given off between the seat of ligature and the sac, as, *e.g.*, in the common carotid.

When the detached portion of clot lodges in the mouth of the sac itself, and so prevents either the ingress or egress of the blood, that blood which remains in the aneurysm forms a passive clot and so fills up the sac. This mode of spontaneous cure is imitated in the treatment of aneurysms by the application of an Esmarch's bandage, the limb being emptied of blood above and below the aneurysm, while the sac of the latter remains filled. The bandage is left on a sufficient length of time to allow of a passive clot being formed before blood is again allowed to circulate. Other methods of spontaneous cure are described, but are certainly of rare occurrence. Thus, the sac is said occasionally to cause pressure on the artery above the seat of aneurysm, and, by thus diminishing the flow of blood, to cause the

formation of a laminated clot. In other cases, again, the development of another aneurysm on the same vessel may cause the circulation in the latter to become so much retarded as to promote a cure. Of this I have myself seen one instance in the case of a woman, who died from an aneurysm of the first part of the right subclavian artery, and in whom a post-mortem examination showed another aneurysm on the third part of the same vessel completely filled with fibrin, and only communicating with the artery by a minute aperture.

Lastly, in some cases inflammation of the sac may cause the formation of clot. This is certainly a very rare event, and the occurrence of inflammation is a thing rather to be dreaded than desired.

In all cases of spontaneous cure, however promoted, there is another factor, namely, the tendency to contraction of the sac and of the surrounding tissues. The latter have been thrust aside by the growth of the aneurysm, and are, so to say, constantly endeavouring to overcome the dilating force and return to their natural positions. Any loss of power in the dilating force of the circulation in the aneurysm is consequently followed by a contraction of the surrounding parts and pressure on the sac itself—a contraction which tends to continue until the former position of the displaced structure is restored.

EFFECTS OF LIGATURE AND OF PRESSURE ON ANEURYSMS.

Allusion has already been made to the mode of cure in the case of distal ligature, but a few words must be said on the results of the Hunterian operation. This operation consists in the application of a ligature to the diseased artery on the cardiac side of the sac. The result of such a proceeding is the occlusion of the artery at the seat of ligature, the diminution of the blood-stream through the aneurysm, and the gradual filling of the latter by laminated clot. The clotting in the sac extends to the artery, and

thus the latter is occluded in two places —at the seat of ligature, and where it formerly communicated with the sac. Between these two points the vessel in most cases remains pervious, and this is always the case where any length of vessel intervenes, and where branches are given off between the ligature and the aneurysm. Where the ligature is applied close above the sac, the clotting may extend along the intervening portion of artery. The application of pressure promotes the cure of an aneurysm by diminishing the blood-stream, just as does a ligature, but the vessel is not obliterated where the pressure is applied.

The changes that occur in the aneurysm after it has been filled by clot may be briefly described. The clot becomes in part organised and is in part removed. The aneurysm becomes very dark and fibrous, and shrinks rapidly. Some years after cure, but little may remain of the original swelling beyond a small oval mass of fibrous tissue. This, together with other points in the cure of aneurysm, is well shown in the accompanying drawing of the arteries of the lower extremity of a man whose superficial femoral had been ligatured five years before death.

FIG. 55.

The Femoral and Popliteal Vessels, five years after ligature of the superficial femoral for the cure of a popliteal aneurysm. The vessel has been occluded by the ligature at A, and again at the seat of aneurysm. The aneurysm itself is represented by a small mass of fibrous tissue, B. The artery between the seat of ligature and the sac is patent.

The aneurysm is converted into a small fibrous lump which encloses within it the remains of the popliteal artery; the latter has been occluded for a length of about 3 inches. The superficial femoral has been occluded by the ligature, but between the seat of operation and the sac the vessel is pervious. (See Fig. 55.)

The chief **dangers** of ligature for the cure of aneurysm are three in number, namely, secondary hæmorrhage, gangrene of the limb, and suppuration in the sac. With regard to the first there is nothing special to say, but the remaining two require some explanation.

In some cases the gangrene is dry, but in many it is moist. The dry gangrene is certainly the result of the arrest of the arterial circulation, but the moist gangrene evidently must be due to some occlusion of the veins as well. This occlusion is sometimes caused by an injury to the main vein at the time of operation, with the consequent formation of a thrombus. In other cases it seems to be due to pressure on the vein by the aneurysmal sac. It is of course a common thing for the main vein to be compressed to some extent by an aneurysm, yet as a rule the venous current is not completely obstructed. As a result of ligature, however, the conditions are altered, for as the *vis a tergo* is much diminished, the force of the venous current is lessened, and also as the sac becomes filled with clot it causes greater pressure on the vein. For these causes, therefore, gangrene after ligature is liable to be of the moist variety. In any case, it commences at the periphery, but whilst sometimes very limited, in other cases it quickly spreads.

Suppuration in the sac is of rare occurrence. It results from the presence of a large amount of clot, which, acting as a foreign body, or, perhaps, becoming infected by micro-organisms, sets up irritation in a few cases, and in some causes the formation of pus. Suppuration of the sac is an event of comparatively late occurrence, and is met with from three to eight weeks after ligature. If the sac be

incised, there is usually no large hæmorrhage, for the same abundant formation of clot which caused the suppuration usually occludes the artery as well.

CIRSOID ANEURYSM.

Cirsoid aneurysm, or aneurysm by anastomosis, consists of a dilatation and elongation of one or more arteries. The dilatation of the vessels is in some cases tolerably equable, but, usually, each diseased artery presents numerous pouches or saculi. The longer the disease lasts, the more extensive does it become, and vessels at first healthy are gradually involved. In many cases the veins and capillaries become greatly distended, and a fully developed aneurysm by anastomosis shows itself as an irregular pulsating tumour composed of large and tortuous vessels opening directly into one another. When a cirsoid aneurysm has existed for some time, the walls of its constituent arteries become greatly thinned.

The patients in whom this form of arterial disease occurs are mostly young adults. It is usually found upon the scalp and forehead; occasionally it commences in a pre-existing nævus. The chief danger of a cirsoid aneurysm is rupture and hæmorrhage.

CHAPTER XXIX.

DISEASES OF VEINS.

VARIX.

A VEIN which becomes more dilated than is natural is said to be varicose.

The chief **causes** of varix are, mechanical obstruction to the venous circulation, and weakness of the heart's action, combined with general debility and loss of muscular tone. In addition to these, certain occupations and visceral diseases promote the development of varix in special localities.

The chief **situations** of varix are the leg and thigh, especially on the inner side, the anus and rectum, and the spermatic cord. In the lower extremity varicose veins are liable to be induced by any occupation which entails much standing, and so favours gravitation of the blood and increased pressure on the walls of the vessel which contain it. The superficial veins are more often diseased than the deep ones, for they are not supported by the muscles. The longer the column of blood, the greater will be the pressure on the veins, and for this reason the internal saphenous vein is more liable to varix than the external. The force of the arterial circulation is also diminished at the extremities of the body, and, especially when the heart is acting feebly, the arterial stream is not sufficiently vigorous to support and propel the venous blood. The constriction of the thigh by tight garters, and the pressure of the gravid uterus on the iliac veins, are also well recognised causes of varix of the lower extremity.

Varix of the veins of the spermatic cord, or "varicocele," occurs much more frequently on the left side than on the right, and commonly originates about puberty. Its causes are supposed to be the length of the column of blood in a vein without valves, the tortuous course and frequent anastomoses of the veins near the testis, and the slight support afforded by the loose and yielding textures of the scrotum. The pressure of the rectum or sigmoid flexure is generally considered to account for the prevalence of varicocele on the left side, but the greater length of the left spermatic vein is a consideration of at least equal importance.

Varicose veins in the rectum, or hæmorrhoids, are specially caused by any obstruction to the circulation through the liver; by constipation, and consequent pressure on the rectal veins by scybalous masses; by the pressure of the gravid uterus, of uterine tumours, or of an enlarged prostate; and by sedentary occupations.

The anatomical changes in varicose veins are easily appreciated. On account of inability to support the blood-pressure, the vessel dilates, sometimes in its whole length, but more frequently at certain places where the pressure is greatest, *e.g.*, behind a valve or at the point of entrance of a branch vein. As the dilatation increases, the valves cease to act efficiently, and thus, when they are most needed to break and support the long blood-column, they gradually become useless, and after a time, may almost completely atrophy. In some cases the vein-walls are thickened to resist the excessive pressure, the thickening being chiefly due to increase of fibrous tissue; in other cases, unable to withstand the dilating force, they become extremely thin and lacerable.

It is commonly supposed that, in the lower extremity, it is the superficial veins alone that become dilated; but this is not the case, for, in almost all patients in whom the superficial veins are varicose, the deeper ones are similarly affected, and the most prominent bulgings often mark the site of a communication between a deep and a superficial

branch. But not only does a varicose vein increase in diameter; it also increases in length, and, in consequence, becomes curved and tortuous. This tortuous condition, in turn, causes still further obstruction to the blood-stream, and at the convexity of each curve, where the obstruction is greatest, pouches of large size are formed.

Effects of varicose veins.—In some instances no material effects result from varicosity of the veins, but in most cases of long standing the tissues which they drain suffer to a greater or less extent. One of the first effects of varix is congestion and swelling of the peripheral parts, with exudation of serum from the distended vessels and increased succulence of the soft tissues.

Sometimes matters go no farther than this, but very commonly the exudation increases and catarrhal inflammation of the skin, or eczema, ensues. After this, ulceration may follow, for the sodden and thinned epidermis is readily destroyed by the most trivial injury, and then the sensitive papillary layer of the skin is exposed. Such exposure results in inflammation, which rapidly progresses to the formation of an ulcer, and this, once formed, will tend to progress so long as the abnormal conditions of the circulation which produced it continue to act.

Another result of varicose veins is atrophy of the tissues in contact with them from the constant pressure of the dilated vessel. In this way the superjacent skin may become extremely thinned, so that in exceptional cases but slight injury is sufficient to rupture the dilated and exposed vein. Profuse hæmorrhage may thus be caused, and, although readily arrested by pressure, a varicose ulcer may originate at the site of the rupture.

The thinness of the skin and the slowness of the blood-stream in varicose veins also predispose to phlebitis and thrombosis, subjects which are dealt with below. It is to attacks of inflammation that most of the more troublesome and painful symptoms of varicose veins are to be attributed rather than to their mere dilatation and tortuosity.

The discoloration of the skin in the neighbourhood of a varicose vein is the result of an escape of some of the red blood-cells and their subsequent disintegration. It is liable to occur in any tissues which are chronically inflamed.

THROMBOSIS AND PHLEBITIS.

Thrombosis is a coagulation of blood in a vessel, the resulting coagulum being called a **thrombus**.

Phlebitis is inflammation of a vein, and is frequently associated with thrombosis.

Coagulation indicates destruction of some at least of the normal constituents of the blood, and is the constant sequel of withdrawal of the blood from the vessels which naturally contain it, unless some special precautions are taken to prevent its occurrence.

Clotting of the blood is supposed to be the result of the destruction of the blood corpuscles, and the freeing from them of a substance which promotes the formation of fibrin from the blood plasma. So long as the blood is contained within vessels in a natural condition, there is little or no tendency to destruction of the corpuscles, and thus it may be said that contact with normal endothelium is sufficient to keep the blood in a fluid state.

The following are some of the chief **causes** of thrombosis :—

First, **retardation of the blood-stream**.—If the flow of blood be retarded in any way, coagulation is likely to result. In wasting diseases, such as phthisis, the feeble circulation sometimes results in the formation of a thrombus in the vessels of the extremities, where the circulation is naturally most difficult. The blood clots first behind the valves, the blood-stream being too feeble to force them open. In other cases, the pressure of a splint, a bandage, or other mechanical appliance may produce clotting at the seat of pressure. The slowness of the blood-stream in varicose veins, already described, is another fertile source of thrombosis.

It is probable that retardation of the circulation causes thrombosis by itself producing an abnormal condition of the wall of the vessel. The latter thus ceases to exercise its normal control over the flowing blood, and on the death of the corpuscles coagulation ensues.

Secondly, **injuries**.—Any injury to a vein may cause thrombosis, but those which lay open the cavity of the vessel are more likely to do so than are mere contusions. Lacerated and jagged wounds are followed by more clotting than are clean incisions.

Thirdly, **contact with foreign bodies** or with tissues uncovered by endothelium.

In the case of the arteries, it has already been mentioned that thrombosis is likely to follow on destruction of the endothelium by atheroma. Atheroma is very rarely met with in the venous system, but the extension into a vein of a new growth, or the presence of foreign bodies, introduced from without, such as sutures, results in coagulation.

Fourthly, **certain constitutional conditions**.—In gout, typhoid fever, pregnancy, and in many of the various forms of blood-poisoning there is a tendency to the formation of thrombi. Whether this is the result of some alteration in the blood itself or of some change in the vessel-wall is not certain.

Fifthly, **extension of inflammation** from surrounding parts, and the consequent causation of phlebitis.

PLASTIC PHLEBITIS.

All cases of thrombosis were formerly supposed to originate in phlebitis, but whilst it is now quite certain that this is not true, there has been too great a tendency to believe that plastic phlebitis does not exist. It would certainly be more than strange if one of the tissues of the body was found to be unassailable by a process which spares none other, and as a matter of fact, we find that veins, like other structures, are liable to inflame.

As far as can be ascertained at present, however, unless phlebitis is caused by such constitutional conditions as gout, typhoid fever, &c., already mentioned as causes of thrombosis, it always owns a local origin.

The following causes may result in inflammation of a vein :—

First, **injuries**.—If a vein be cut across, as in an amputation, it, like all other injured structures, becomes the seat of a local inflammatory process which results in the exudation of lymph, both into the walls and into the cavity of the vein. This lymph usually undergoes organisation into fibrous tissue in exactly the same way as in arteries which have been injured. If the injury has been a clean cut, not completely dividing the vein, the resulting scar will be imperceptible. Contusions and lacerated wounds are also liable to cause local phlebitis, and continuous pressure may produce a like result.

Secondly, **extension of inflammation** from surrounding parts.

In all cases of inflammation, the veins share in the changes which occur in the parts around them. Exudation of cells, vascularisation of the inflammatory products, softening of the vein-wall, and finally destruction by suppuration, may all ensue. If the inflammatory exudation takes place beneath tense structures, such as fasciæ, the pressure of the exuded material is liable to give rise to a widespread thrombosis, in addition to the clotting which results from the inflamed state of the vein-walls.

Thirdly, **presence of a thrombus**.—A thrombus in a vein causes a certain amount of irritation, and inflammation of the vein-wall ensues. The termination of this phlebitis will depend upon the character of the clot; suppuration occurs only when the latter is in a septic condition.

The changes that occur in the vein as the result of phlebitis are such as are common to all inflammations, and need no special description. They include swelling and cell exudation and proliferation, and the cells may either be ab-

sorbed, or may remain and cause a permanent thickening of the vein-wall, by the formation in it of fibrous tissue. The effect of the phlebitis on the circulating blood is the formation of a thrombus, and the consequent blocking of the affected vein. This may certainly occur independently of exudation of lymph into the cavity of the vessel, and results from the injury done to the endothelial lining by the inflammatory process.

It will thus be seen that whilst, on the one hand, thrombosis may cause phlebitis, on the other, phlebitis results in thrombosis. Further, the same causes which produce thrombosis may also cause phlebitis, and in many cases it is quite impossible to say whether the thrombosis is primary or whether it is secondary to an antecedent phlebitis.

When a thrombus has been formed in a vessel, it may extend either with or against the blood-stream, and frequently it grows in both directions. The blood on each side of the thrombus is not in a stagnant condition, but, on the contrary, is kept in a state of constant and regular agitation. Consequently, the thrombus increases not only by a gradual extension of coagulation through a column of stagnant blood, but by the separation of fibrin which results from the constant movements of the fluid.

If the clot extend towards the heart, its formation is usually arrested when it reaches the orifice by which the occluded vein opens into a larger trunk, a termination which is promoted by the greater vigour of the circulation in such a vessel. Such an arrest is not, however, of constant occurrence, for it occasionally happens that clot or fibrin is deposited upon the thrombus where it protrudes into the main trunk, and by a continuance of this process the latter may itself be occluded.

When the clotting progresses against the blood-stream, it does not usually extend beyond the first patent collateral branch. If the original thrombosis has commenced in a main vein, such as the femoral, then, on account of the

great interference with, and the retardation of, the blood-stream, clotting is liable to be very extensive.

Changes in the thrombus.—The first and most constant change is **contraction.** This contraction is always very noticeable in a blood-clot formed outside the body, and, for long after the formation of the coagulum, serum continues to be squeezed out. Exactly similar changes occur in the thrombus formed in a vein, and, in consequence, the wall of the vessel is puckered at the places where the plugged branch veins enter it, although when examined post-mortem the vein as a whole is much more distended and prominent than is a healthy vessel. All thrombi are more or less adherent to the vein-wall. In most cases the adhesion is simply by coagulated fibrin, but is occasionally, in old clots, by fibrous tissue.

After it has contracted, the thrombus becomes gradually decolorised, the red blood-cells disintegrating, and their colouring matter being diffused and removed in the manner already described in the chapter on "Contusions." The older a clot, the whiter it is.

In very many cases the clot is finally **absorbed** in great part, if not entirely. It is most probable that this absorption is to a large extent the result of simple degenerative changes which occur in its more central parts in consequence of insufficient nutrition; but there can be little doubt that much of the absorption is due to the action of leucocytes which are exuded into the clot from the wall of the vein in which it lies. It is a well-ascertained fact that the removal of clots outside the vessels is due to the action of white blood-cells, and, as the number of leucocytes in a thrombus constantly increases from the time of its first formation, it is highly probable that here also they are employed in a similar work.

When absorption does not take place, **organisation** may ensue. The process is the same as that already described as occurring in the internal clot in arteries after injuries, and is the result of a proliferation of the endothelial and sub-

endothelial cells, and their development into fibrous tissue. This fibrous tissue afterwards contracts, just as does a scar, and may either draw the vein-wall with it, or may shrink to one side of the vessel and so allow of the re-establishment of the circulation. Much more rarely the blood bores a hole through the organised clot, and thus, as it is termed, "tunnels" the thrombus.

Lastly, thrombi which are of long standing may undergo calcareous degeneration and form "phleboliths." These small concretions are very common in the veins of the prostatic plexus in old subjects.

Results of thrombosis.—The result of the arrest of the circulation in any vein will depend on the freedom with which the blood is able to return by other channels. Where the occluded trunk is small and superficial, the tissues which it drains may present no change. If, on the contrary, the vein be the main one of the limb, *e.g.*, the femoral, then there is, at first, a blueish discoloration with swelling, followed, after a time, by a dead-white colour and a solid sort of œdema, with alteration in the shape of the leg and thigh. When the vein is permanently blocked, this œdematous condition may persist for years, and is generally attended by much interference with the usefulness of the part.

Rarely, portions of clot become detached, and are carried by the circulation to the right side of the heart, and thence to the lungs. The effects of this embolism of the branches of the pulmonary artery depend almost entirely on the size of the embolus, and the consequent interference with the pulmonary circulation.

SUPPURATIVE PHLEBITIS.

Suppurative phlebitis is always the result of septic changes in the tissues around the vein, and is usually the result of a wound. There is no such thing as idiopathic or primary inflammation of a vein, with exudation of pus into its cavity.

Suppurative phlebitis, indeed, is usually accompanied by

diffuse cellulitis, an inflammation which readily extends along the cellular tissue which surrounds the venous trunks. There is thus a so-called "periphlebitis," and the inflammatory changes secondarily extend to and implicate the vein-wall itself. In consequence of the alteration thus produced in the lining membrane the blood clots, and the vein is plugged. The inflammation progresses, and the vein-wall is involved in the suppuration. Finally, in parts, the coats of the vein are destroyed, and pus now mingles with the clot already formed. Hæmorrhage does not occur, and the pus does not mingle with the blood, for the reason that clotting always precedes the extension of suppuration into the vein. In suppurative phlebitis, the clot never becomes absorbed or organised, but is always destroyed with the vein in which it lies. Circulation, therefore, is never re-established in the vein itself.

There is another way in which suppurative phlebitis may originate. In cases of pyæmia, the clots which are formed in the veins become themselves impregnated with infective material absorbed from the wound. These clots excite suppurative inflammation in the vessels which contain them, and the clots themselves become disintegrated and mingled with the products of inflammation. If portions of these broken-down and septic clots are carried into the circulation, not only will they cause embolism of the pulmonary vessels, but wherever they lodge they will infect the tissues in which they lie, and will excite in them an inflammation similar to that which was in progress in the part from which they were originally derived. (See Pyæmia.)

CHAPTER XXX.

EMBOLISM.

Embolism is the term applied to the plugging of a vessel by a foreign body—usually a blood-clot—which has been carried from a distance by the blood-stream. The foreign body is called an embolus.

An embolus may be formed in either the arterial or venous system ; in the left or right side of the heart. Most commonly it is formed in the left side of the heart, and consists of fibrin or blood-clot from diseased aortic or mitral valves. In some cases these emboli contain calcareous material. An embolus originating in the heart will, when swept into the blood-stream, be carried into the systemic circulation, and will be arrested when it comes to a vessel too small to allow its transit. Very commonly the arrest occurs above a bifurcation.

In other cases the embolus originates in a vein ; and then consists of clotted blood. If a portion of clot in a thrombosed vein be detached, it will be carried to the right side of the heart, and thence into the pulmonary circulation, where it will plug some branch of the pulmonary artery.

The immediate result of the lodgment of an embolus is the partial or complete arrest of the blood-stream. Very shortly, the blood clots at the seat of embolism, and a thrombus is thus formed around the embolus. The presence of the embolus excites inflammatory changes in the vessel-wall, and from the vasa vasorum of the latter new vessels are developed, which penetrate the embolus and its surrounding clot. Inflammatory changes now occur, and

the clot, becoming "organised," adheres to the vessel-wall. In this way the plugged artery is permanently occluded.

Such is the usual result of embolism, but, in rare cases, the embolus, being composed of soft clot, may be disintegrated, and the channel of the vessel may be reopened. In other, and still more rare, instances, an aneurysm may form at the seat of embolism, as described in the chapter on Aneurysm.

In addition to the local effects of embolism, the parts to which the plugged vessel is distributed suffer from deficient supply of flood. The moment that the artery is occluded, the patient usually suffers severe pain at the place where the embolus lodges, while in the peripheral parts he experiences sensations of numbness, with superficial burning pain and loss of power in the muscles. Unless the collateral circulation enlarges, the most distal parts will gradually pass into a condition of dry gangrene, and, in some cases, such a result is hastened by the detachment of small portions of the embolus or its surrounding thrombus and consequent embolism of other and more distal vessels. (See Gangrene.) In many cases of embolic gangrene, complicated by heart disease, the patient does not long survive the death of a limb.

The effects of embolism of a branch of the pulmonary artery depend to a great extent on the size of the occluded vessel. If very large, death may ensue almost instantaneously; if not, recovery may follow after temporary consolidation of the lung around the plugged vessel. The subject of "septic embolism" has already been dealt with in the chapter on Pyæmia.

Instead of blood-clot and fibrin, an embolus may be composed of portions of a tumour which has grown into the surrounding vessels without causing their obliteration, of micro-organisms, of fat, of parasites, &c. Such cases are necessarily of considerable rarity, and, with the exception of fat embolism, of no great practical importance. **Fat**

embolism is of some interest, for it seems to explain cases of death after both simple and compound fractures which would otherwise be obscure. It results from the destruction of the fat-cells in the fractured bone and the passage of fat-granules—often aided by the tension produced by inflammatory exudation—into the lymphatics and veins. From these vessels the fat is carried to the lungs, and causes obliteration of the pulmonary capillaries.

CHAPTER XXXI.

DISEASES OF THE LYMPHATIC SYSTEM.

LYMPHANGITIS.

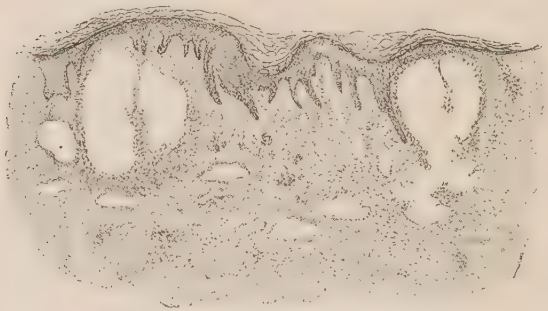
Lymphangitis, or inflammation of the lymphatic vessels, is commonly the result of the transmission by them of irritating material from a wounded surface. It is especially prone to follow poisoned wounds of all kinds, more particularly those in which the poison is a bacterial one, and is thus frequently seen in cases of post-mortem or dissection wounds, as well as in the injuries sustained by butchers from implements used in their work. In many cases the injury is extremely slight, such as a scratch or a prick, and the retention of pus even in minute quantities seems to greatly favour the development of the disease. Commencing in the radicles of the lymphatic system, the inflammatory process extends along their cellular-tissue sheath as well as in the vessel-wall itself; when the trunk is a superficial one, the skin commonly shows a red line in the course of the affected lymphatic. As a result of the inflammation, the vessel-wall becomes thickened and its contents turbid or coagulated. Sometimes suppuration ensues, and very often lymphadenitis complicates the course of the disease.

LYMPHANGIECTASIS AND LYMPHORRHŒA.

Lymphangiectasis, or dilatation of lymphatic vessels, results from anything which obstructs the flow of lymph to

such an extent that the collateral as well as the main channels are closed. This condition sometimes results from a chronic lymphangitis, but more often from the pressure of some new growth. In not a few instances no cause can be assigned. The common situations for varicose lymphatics are the inguinal regions and the inner side of the thigh. The appearance of the skin in such cases has been likened to the rind of an orange, and when numerous lymphatics are much dilated they present just the same tortuous outline, with pouches at the curvatures, as do varicose veins. They are, however, quite colourless, and only covered by the surface epithelium. Occasionally, in

FIG. 56.



Lymphangioma from the Skin of the Buttock. The dilated lymph spaces causing protrusion of the surface epithelium are well seen. (Crouch, 1 inch.)

connection with this disease, there is a rupture of the dilated vessels and a discharge of lymph—a condition known by the name of **lymphorrhœa**.

Lymphangioma is merely an exaggeration of lymphangiectasis, the dilated lymphatics forming definite swellings or tumours. These growths are sometimes congenital, and I have seen one on the buttock of a boy which looked like a diffuse papilloma, with very translucent papillæ. Microscopically, the growth is seen to be composed of dilated

lymphatics. In cases of macroglossia also, much of the enlargement is found to be due to dilated lymph channels.

ELEPHANTIASIS.

The term elephantiasis is applied to all all forms of chronic hypertrophy of the skin and subcutaneous tissue which result in the production of much deformity. The term "spurious elephantiasis" has been lately applied to those cases in which the disease is the result of inflammation of no specific or definite form. Good examples may be seen in the enlargements of the legs of patients with chronic ulcers, or of the scrotum in cases of long-standing urethral stricture with fistulæ. The enlargement is due to an overgrowth of the connective-tissue elements of the parts. This increase is due both to the vascularity of the chronically inflamed tissue and the attendant exudation, and also to the subsequent interference by pressure with the flow of lymph in the lymphatics.

The term **true elephantiasis**, or **elephantiasis arabum**, is applied to a special form of the disease which is endemic in certain countries—notably certain parts of Asia, Arabia, and Barbadoes—and which develops independently of previous local inflammations. The natives of the countries infested by the disease are more liable to attack than are Europeans, and it is supposed by some writers that the want of clothing on the legs and feet renders these parts more susceptible. The onset of the disease is marked by fever, and the skin of the affected part—generally the leg or scrotum—becomes red and inflamed, with small vesicles on the surface. These attacks of cutaneous inflammation frequently recur, and each one leaves in its train a permanent distension of some of the lymphatics. The subcutaneous tissue is at first soft and œdematous, and pits on pressure; after a time, it becomes hard and brawny, and the skin is either covered with vesicles and dilated lymphatics, or, in the later stages is eczematous, roughened, tubercular, and horny. The amount of newly formed

fibrous tissue is frequently enormous, the scrotum weighing as much as one hundred pounds or more, and the thigh measuring one or two feet in diameter.

The cause of true elephantiasis has long been the subject of much speculation, and it has been attributed to exposure to excessive heat, to the absorption of poison by the skin, to an inherited predisposition, &c. It is very generally believed at present that the disease is the result of the passage of a nematode worm (the filaria sanguinis hominis) into the lymphatic tissues. The adult female worm is about $3\frac{1}{2}$ inches in length, and as thin as a very fine hair, having a breadth of about $\frac{1}{100}$ of an inch; the adult male has not yet been obtained entire, so its length is uncertain, but in breadth it is about $\frac{1}{100}$ of an inch.

The worm breeds freely in the human body, and appears to choose for its habitat some of the lymphatic tissues, just as the trichina selects muscle. The embryos, which are about $\frac{1}{70}$ of an inch in length, and $\frac{1}{250}$ in width, are endowed with free mobility from their birth, exhibiting lashing, eel-like movements when placed under a microscope. They readily make their way along the lymphatic vessels, and reaching the thoracic duct, enter the blood-stream. In the blood of an affected person they are usually entirely absent during the day, beginning to appear about evening and being present in enormous numbers during the night.

This periodicity depends on the wakefulness or otherwise of the patient, for if sleep be taken during the day instead of at night, the filariæ also change their habits. Whither they disappear during the waking hours is not known, and it has been suggested that none of them live more than a night, and that each evening a fresh brood is produced. These embryo filariæ do not undergo further development whilst in the human body, but it appears from the investigations of recent observers—especially Manson—that the mosquito is their next host. They are taken up by the mosquito when the latter draws blood at night, and in it they further develop, cause the death of their

host, and then escape into the water near which these insects usually live. From the water they may gain access to man either by the alimentary tract or through the skin. There is yet some doubt in which of these ways, but the former appears much the more probable.

The presence of filariæ is supposed to produce elephantiasis by keeping up a chronic irritation and inflammation of the lymphatics in which they breed, but it is also suggested that the lymphatic vessels themselves are occasionally mechanically blocked by prematurely discharged, immature ova which have a much greater transverse diameter than the embryo filariæ. It may be mentioned that other diseased conditions are also ascribed to the presence of the filaria sanguinis hominis, especially some forms of endemic hæmaturia, chyluria, and hydrocele containing chylous fluid.

LYMPHADENITIS.

Lymphadenitis, or inflammation of a lymphatic gland, is generally the sequel of an injury to, or inflammation of, some peripheral part in which the lymphatic vessels passing to the inflamed gland arise, and is often a complication of lymphangitis. In some cases direct injury appears to act as the exciting cause; in others, excessive or long-continued exercise may start the affection. Inflammation of lymphatic glands is also met with in various forms of specific disease, *e.g.*, gonorrhœa, erysipelas, &c., but at present only the simple non-specific inflammations will be considered.

The affected gland presents all the ordinary appearances common to inflammations in general; it becomes vascular, soft, and swollen, and the loose connective tissue in which it lies shares in the pathological changes. On section, it is found to contain a good deal of fluid, and microscopical examination shows that it is infiltrated with leucocytes, which are especially numerous in the cortex. Lymphadenitis may terminate in different ways. In favourable

cases resolution occurs, the vascularity subsides, and the exuded leucocytes return to the lymphatic channels. Very commonly suppuration results, the leucocytes increase in number, many of them die, and small collections of pus form in different parts of the gland and in the surrounding cellular tissue. These collections run together, and an abscess forms and comes to the surface. In severe cases the inflamed gland sloughs, and the abscess which necessarily occurs in such cases exposes, when it bursts, the necrosed gland lying in its floor. Where the cause of the original inflammation is itself persistent the inflammation becomes chronic, the cells, as in all chronic inflammations, tend to develop, and the affected gland and the neighbouring tissues are enlarged and indurated by the growth in them of connective tissue. If the patient be strumous, tubercle may develop, and the inflammatory products will then caseate or calcify in the manner already described in the chapter on Struma.

TUMOURS OF LYMPHATIC GLANDS.

Lymphoma.—A lymphoma is a tumour composed of lymphatic glandular tissue. Its most common situation is the neck, and it may affect one or more glands. Lymphomata vary in size, and although it is seldom that any single gland attains a greater size than that of a hen's egg, several affected glands may together form a mass of considerable bulk. These growths are encapsuled, do not infiltrate the neighbouring tissues, and although often rather adherent, do not show signs of inflammatory thickening. The tumours are not malignant and are painless. On section, except for their unusual size, they present the appearance of a normal lymphatic gland. Microscopically, they do not differ from lymphoid tissue, and it is difficult to say whether they should be classed amongst the simple hypertrophies or the new growths.

Lymphadenoma.—Lymphadenoma is a disease characterised by overgrowth of lymphatic glandular tissue in

many parts of the body. The growth is most common in the lymphatic glands themselves, but is found also in the medulla of bones, the intestinal canal, the spleen and the connective tissues. As regards the lymphatic glands the new growth is in some cases soft and almost brain like (**soft lymphadenoma**): in others hard, elastic, and tough (**hard lymphadenoma**). In the first variety, contiguous diseased glands not infrequently cohere, and also become closely adherent to the surrounding tissues. The growth is in many cases very rapid, and neighbouring parts may be either compressed or infiltrated. On section, the diseased glands present a greyish or greyish-white tint, mottled in places with red. Microscopically examined, the growth is seen to be composed, first, of round cells, similar to those of a lymphatic gland, and lying in a delicate matrix of connective tissue arranged in the form of a network; and secondly, of large, round, or oval multinucleated cells, similar to those of sarcoma, with which there may be mingled a certain number of spindle-cells. These growths are often called **lympho-sarcomata**, and there is no hard-and-fast line between lymphadenoma and lympho-sarcoma. Their presence is frequently accompanied by great debility, anæmia, and exhaustion, with emaciation, diarrhœa, and vomiting. They may cause death in this manner or by pressure on import and viscera. Their development is often associated with secondary growths in the viscera and in the bones.

The hard lymphadenomata are not so malignant as is the soft variety, being slower in their growth, more encapsuled, and less widely distributed. The disease tends to extend along the several glands, forming a lymphatic chain, rather than to affect many different groups or to occur in the viscera. In structure the hard lymphadenomata differ from the soft variety in the greater abundance of their fibrous stroma—a condition which accounts for their greater firmness. Between the hard and soft lymphadenomata transitional forms may be found.

Leucocythæmia.—In this disease we have a large increase in the number of white corpuscles of the blood and enlargement of the lymphatic glands or spleen, or of all the lymphoid structures, frequently with marked changes in the bone marrow and secondary deposits in the viscera. A fatal termination is very common.

Leucocytosis.—This term is used to indicate a simple increase in number of the white blood-cells. It is a comparatively common complication of lymphadenoma, but occurs also in many other morbid conditions.

Sarcoma.—Sarcoma of a lymphatic gland occurring as a primary growth, not of the variety already described as lympho-sarcoma, is of rare occurrence. Such tumours are, however, occasionally met with, and I have seen several such cases affecting the axillary glands. In some the growth was composed of large, in the others of small, round cells. Other varieties have been described.

The frequency with which the lymphatic glands are secondarily affected by malignant tumours is well known, and is mentioned in connection with the new growths of different organs.

CHAPTER XXXII.

DISEASES AND INJURIES OF THE LARYNX.

Simple acute laryngitis, or inflammation of the larynx, occurs most commonly as a result of exposure to wet or cold; but may result from extension of inflammation from neighbouring parts, from inhalation of irritating chemical vapours, and from injuries inflicted by foreign bodies or by hot liquids; it is also, but more rarely, met with in some of the exanthemata, and especially in measles.

It is characterised by acute hyperæmia and redness, with some swelling and the formation of a watery or viscid discharge. In severe cases the inflammation may terminate in the formation of an abscess or of ulcers, whilst very exceptionally the mucous membrane may even slough. Small ecchymoses are comparatively common, and the term "hæmorrhagic laryngitis" is sometimes employed to indicate cases where the expectoration is blood-stained.

Œdematous laryngitis is but a variety of the acute form, in which œdema is developed to an unusual extent; it is most commonly seen in connection with scalds, and the extension of erysipelatous inflammation.

Simple acute laryngitis is seldom fatal in adults, but in children, in whom the glottis is but a narrow aperture, dyspnœa is common on account of the obstruction caused by swelling, and is sometimes very urgent; it is increased at intervals by spasm or by the collection of viscid mucus. In œdematous laryngitis dyspnœa is, in adults as well as in children, a most prominent symptom, and is caused by the

great swelling of the ary-epiglottic folds and ventricular bands. Dysphagia and the sensation of a foreign body in the throat are also often complained of and result from the swollen state of the epiglottis, which is rendered rigid and erect by the infiltration of fluid beneath its mucous membrane. In even the most severe cases the œdema is limited to the larynx, and does not extend below the true vocal cords.

Œdema of the glottis occurs also as a complication of all the other varieties of laryngitis, and is sometimes met with in general dropsy and in diseases of the kidneys.

Simple chronic laryngitis is commonly the result of excessive use of the voice, but occurs also as a sequel to acute laryngitis, and is often met with in patients who are habitual drunkards. In such cases the vocal cords become red, thickened, and fleshy, and the whole mucous membrane is usually irregularly thickened by the growth of newly formed fibrous tissue in its substance, whilst enlargement of the mucous follicles and destruction of the surface epithelium are occasionally seen. The term "follicular laryngitis" has been applied to those cases in which the follicles are especially enlarged and distended with mucus. Small circular ulcers sometimes form in this variety of laryngitis.

CROUPOUS AND DIPHTHERITIC LARYNGITIS.

The term croup was for long used to indicate an inflammation of the air passages associated with the formation of membrane, and in this sense it is still frequently employed. At the present time, however, it is believed by an increasing number of observers that cases of so-called "croup" are either examples of diphtheria or of simple laryngitis, and my own experience, derived from a considerable number of post-mortem examinations as well as from clinical practice, has convinced me that laryngitis with the formation of membrane does not exist as a disease separate from diphtheria.

Cases of laryngeal dyspnœa occurring in children may therefore be looked upon as either instances of simple

laryngitis with associated spasm of the muscles of the glottis, or if membrane is present, as of a diphtheritic nature.

Diphtheria is a constitutional disease characterized by an inflammation of the fauces and air-passages with the formation of membrane.

The disease is due to a specific organism, and results from bad hygienic conditions. It is most common in children, and is more fatal in them than in adults. It commonly commences in inflammation of the tonsils, uvula, and pharynx, and in most cases the inflammatory process does not extend to the larynx at all. In some of the worst cases the diphtheritic inflammation is confined to the nasal mucous membrane.

The affected parts are at first red, swollen and inflamed, but soon assume an ashen-grey tint, and after a day or two become covered with a thick leathery membrane. If this is peeled off a raw granulating surface is exposed. The microscope shows that the mucous membrane is covered with dense masses of bacilli and micrococci, and that there is cell exudation into its deeper parts. The membrane is generally formed by the necrosis or death of the epithelial cells and the upper part of the sub-epithelial tissue, so that it may be said that in diphtheria there is an inflammation followed by sloughing of portions of the affected mucous membranes. If the slough or membrane is peeled off, it of course exposes a granulating surface, and when the membrane has been separated the resulting ulcers cicatrize. Diphtheria is a very fatal disease, and may cause death in several ways. In bad cases the patient may die of the general constitutional affection before any membrane has been formed. In other cases, death results from extreme anæmia and exhaustion, but in many patients, and especially in children, death ensues from implication of the air-passages and dyspnœa. This may be due to the swelling of, or formation of membrane upon, the mucous lining of the larynx, but in other cases is the result of an extension of the membrane to the trachæa and the bronchi, and in not a few instances is due to the development of bronchitis and broncho-pneumonia.

In most cases of diphtheria there is an associated enlargement of the cervical lymphatic glands and albuminuria. Later on, diphtheritic paralysis may develop.

Tubercular laryngitis is almost invariably a sequel to tubercular disease of the lungs, and is said to occur in about 30 per cent. of cases of tubercular phthisis.

It commonly commences with a general œdematous appearance of the larynx, the mucous membrane of which is often abnormally pale, the parts most early affected by the swelling being the arytenoids, with the neighbouring mucous membrane, and the epiglottis. In this swollen membrane a microscopical examination shows the deposit of tubercle, and very quickly the smooth epithelial lining becomes first raised in small swellings, and subsequently pitted with minute ulcers, which as they increase in size coalesce and extend in depth, and rapidly destroy the epithelial surface. The ulceration often commences near to the posterior attachment of the vocal cords, but subsequently may extend to any part of the larynx. In exceptional cases the ulceration results in perichondritis and destruction of the laryngeal cartilages. Dyspnœa is not generally a prominent symptom, but in exceptional cases may be severe enough to warrant the performance of tracheotomy.

Syphilitic laryngitis occurs under several forms. In secondary syphilis the larynx is often attacked by an apparently simple inflammation with redness and catarrh, whilst superficial ulceration, and much more rarely mucous tubercles, may develop.

In late syphilis the larynx may be affected by deep and rapidly extending ulceration, which is very liable to penetrate and attack the cartilages; it is rarely the result of the breaking down of gummata. In bad cases there may be caries or necrosis of the cartilages, with subsequent extension of the inflammation into the subcutaneous tissues and the formation of abscesses. In all cases the epiglottis is especially liable to be attacked and may be completely destroyed. Occasionally the inflammation commences in

the perichondrium, and only subsequently extends to the mucous membrane. The inflammatory process is liable to be very chronic, but in any case acute œdema may suddenly ensue and urgent dyspnœa may result. In cases of long standing there is often much thickening of the mucous membrane and cicatricial contraction, whilst more rarely

FIG. 57.

Syphilitic Ulceration of the Larynx with formation of cicatricial bands and destruction of the epiglottis.

bands of membrane and adhesions unite the opposed inflamed surfaces. In this way the orifice of the glottis may become much contracted, and permanent dyspnœa and stenosis may result. The larynx may be affected in any of the above-mentioned ways in patients with congential syphilis.

TUMOURS OF THE LARYNX.

The most common innocent tumour of the larynx is a **papilloma**. The papillomata of the larynx differ somewhat

from each other, and may be either single or multiple, pedunculated or sessile. They occur at all times of life, but are rare after fifty, and are relatively common in childhood. Their most frequent seats are the vocal cords, especially their most anterior parts, and the ventricles. Their surface is warty and rough, and their colour grey

FIG. 58.

Papilloma of the Vocal Cords.

or red. The most prominent symptom caused by these tumours is hoarseness of voice, but in children they may cause much spasm and dyspnœa, and occasionally terminate fatally.

Fibromata of the larynx are slowly growing tumours, most common in people of middle age, and usually situated on the vocal cords. They are commonly single, and form round or oval smooth tumours. They are generally no larger than a pea, but may be as large as a hazel-nut, and

are either sessile or pedunculated. Their cut surface is white, and the microscope shows that they consist of dense fibrous tissue.

Mucous cysts are developed in connection with the mucous follicles of the larnyx. They are most common on the back of the epiglottis and in the ventricles, and vary in size from that of a pin's-head to that of a hazel-nut. They contain clear mucus.

In addition to these above-mentioned innocent growths,

FIG. 59.

Epithelioma of the Larynx.

adenomata, angeiomata, enchondromata, lipomata, and myxomata have been described; all these varieties of tumours are, however, of very rare occurrence.

The most common malignant growth of the larynx is undoubtedly **epithelioma;** it may be either intrinsic or extrinsic in its origin. The former term is applied to tumours which commence completely within the laryngeal box or grow on the true vocal cords, in the ventricles,

and on the parts beneath the cords. Tumours originating in the epiglottis or the ary-epiglottic folds are said to be extrinsic.

The patients in whom epitheliomata occur are most frequently men over middle age, and the growth may originate in any part of the larynx. The surface of these tumours is generally very definitely papillary or warty, and for this reason mistakes in diagnosis are common. The growth of epithelioma of the larynx is comparatively slow, and life is generally prolonged for rather more than two years.

When originating inside the laryngeal box the progress of the tumour is much slower than when it commences extrinsically, and the lymphatic glands are not affected until late in the disease. Epithelioma spreads very slowly in cartilage, but as it attacks it perichondritis is set up, and necrosis of portions of cartilage may result. When the growth reaches the extra-laryngeal tissues, suppuration and sloughing commonly ensue, and the tumour may fungate through openings in the skin.

Death often results from septic broncho-pneumonia, set up by the foul state of the larynx, but in other cases is brought about by dyspnœa or exhaustion. The lymphatic glands which are infiltrated sometimes form large breaking-down, sloughing masses, and are more likely to be early implicated in the extrinsic variety. Dissemination and the development of secondary tumours in the viscera are very rare. Epithelioma of the larynx is usually of the squamous-celled variety, but columnar-celled growths have been described. Spheroidal-celled alveolar carcinoma is also but rarely met with.

Sarcoma of the larynx is a rare disease. The tumours are sometimes of considerable size, and are at first smooth and globular, but subsequently ulcerate and infiltrate the surrounding parts in the same way as does an epithelioma. The tumour may be of either the round or spindle-celled variety, and, unlike the epitheliomata, rarely affects the lymphatic glands; dissemination is also uncommon.

INJURIES OF THE LARYNX.

Blows upon the larynx may cause much reflex spasm and dyspnœa, which is sometimes urgent. Fractures of the cartilages are rare, and may result either from a blow or a squeeze; the thyroid cartilage is more often fractured than the cricoid. Such injuries are always serious, and, when the mucous membrane is torn, hæmoptysis and dyspnœa are common. Death not uncommonly results from obstruction to the passage of air by displacement of the fractured cartilage.

Scalds of the larynx are most often seen in children, and commonly result from attempts to drink from a kettleful of boiling water. In these cases the lips, cheeks, and tongue are commonly blistered, white, and swollen, the voice is husky, and swallowing is painful. Œdematous laryngitis is very liable to supervene within an hour or two, and in the absence of treatment death from dyspnœa is of common occurrence.

FOREIGN BODIES IN THE AIR-PASSAGES.

Foreign bodies always obtain access to the air-passages during the act of inspiration, except in cases where the muscles of the glottis are paralysed, as, *e.g.*, after diphtheria; they cannot normally obtain an entrance during deglutition. In most cases they obtain entrance through some sudden inspiratory act whilst the foreign body is in the mouth; thus, a piece of meat may pass into the larynx if the patient suddenly laughs whilst eating, or a pin or other foreign body held between the lips may in a similar way obtain entrance. Foreign bodies may also pass into the air-passages during deep anæsthesia when the normal sensibility of the glottis is numbed.

A foreign body may lodge in the larynx, the trachea, the bronchi, or the lungs, and the symptoms differ according to the position in which it rests.

In the larynx a large mass of meat or similar material may completely block the glottis, and cause death from

dyspnœa in a few minutes; and in other cases, although not large enough to mechanically close the whole glottis, a foreign body may cause death through spasm set up by its irritation. If death does not at once result, and the foreign body remains in the larynx, it interferes with respiration and speech to a varying extent, but the symptoms are continuous so long as it remains, although they are liable to sudden exacerbation at any time. If not removed, the foreign body will set up laryngitis, and death may result either from this or from inflammation spreading to the trachea and bronchi.

If the foreign body passes into the trachea it is never large enough to completely plug that tube, and after an initial attack of dyspnœa the symptoms are liable to subside. As the vocal cords are not interfered with, there is no marked alteration of voice, and respiration may be tranquil. The patient is, however, liable to sudden and severe attacks of dyspnœa; these are caused by the foreign body being coughed up against the glottis, which immediately closes spasmodically, and during such an attack the patient may die of suffocation.

If the foreign body remains in the trachea, death will result either from spasm of the glottis or from inflammation of the trachea and bronchi.

The lodgment of a foreign body in a large bronchus results in the obstruction to the passage of air into a given portion of lung, and is accompanied by symptoms of dyspnœa, which are urgent in proportion to the size of the bronchus which is plugged. Bronchitis and broncho-pneumonia will subsequently ensue, and the lung supplied by the plugged bronchus will become collapsed.

In cases where the foreign body is very minute and passes into a terminal bronchus, the initial dyspnœa is but slight, but further symptoms will ensue in consequence of inflammation and suppuration of the surrounding lung tissue.

CHAPTER XXXIII.

DISEASES OF THE THYROID GLAND.

Inflammation of the thyroid gland apart from other diseased conditions is an event of some rarity, although it is by no means uncommon for a goîtrous gland to become inflamed. The changes that occur are such as are seen in similar conditions of other soft tissues, and the inflammation occasionally terminates in the formation of pus.

Atrophy of the thyroid is a frequent accompaniment of myxœdema, but is otherwise of no importance.

GOÎTRE, OR BRONCHOCELE.

The term goître is applied to any enlargement of the thyroid gland which is not caused by inflammation or malignant growth; its causes have been much discussed, and are not yet satisfactorily settled. In some cases the disease is hereditary, and commonly commences in early life; in a very large percentage of instances it occurs only in certain definite localities, where it is endemic.

The disease is considered by some writers to be caused by any occupation or habit which favours congestion of the veins of the neck, such as the playing of wind instruments. For many years goître has been attributed to drinking water derived from melted snow or ice; but there does not appear to be any truth in this theory. It is also supposed —and with more probability—to be due to the presence of large quantities of lime salts in drinking-water.

Structurally, goîtres may be divided into four classes,

though it must be remembered that between all of these there are connecting links, and that more than one of the morbid conditions may be found in the same growth.

First, **simple hypertrophy.**—In this form there is an overgrowth of the glandular tissue, and in most instances the enlargement is symmetrical, both lobes as well as the isthmus being hypertrophied in proportion to their original size. In other cases localised hypertrophies occur, and definite glandular tumours—**true adenomata**—may develop in the substance of the lateral lobes or in the isthmus. Such growths are usually definitely encapsuled and seldom attain a large size. Whether the growth be universal or local, the new tissue does not differ from that of a healthy gland, being composed of closed vesicles lined by epithelium containing a clear sticky fluid, embedded in a stroma of loose connective tissue.

Second, **cystic goître.**—In cystic goître there is a development of cysts of unusual size. These cysts commonly occur in glands in which there is also some true hypertrophy, but may develop in otherwise healthy thyroids. They are formed by a mucoid or colloid degeneration of the walls of separate vesicles, which are thus thrown into one another, and form cavities of various dimensions. In other cases they appear to result from cystic degeneration of an adenoma. Such cysts are sometimes single, but more often multiple. They contain either clear serous fluid, viscid colloid material, or a dark bloody liquid, with a grumous or coffee-ground-like deposit. They may attain a great size, and sometimes develop with great rapidity as the result of spontaneous hæmorrhage into them. Under such circumstances the pressure of the cyst may cause dyspnœa, or even dysphagia.

Third, **fibrous goître.**—In fibrous goîtres there is an excess of the fibrous stroma as compared with the glandular tissue. The fibrous growth is common in old goîtres, and generally occurs in a gland which has previously been the seat of simple hypertrophy. In other goîtres of long

standing calcareous degeneration is frequently met with, and in some cases the whole thyroid is converted into a calcareous mass.

Fourth, **pulsating goître**.—This is a form of bronchocele in which there is an excessive development or dilatation of the vessels. It is really only a variety of simple hypertrophy. Pulsating bronchocele is sometimes associated with protrusion of the eyeballs.

The effects of a goître depend both on its size and on the rapidity of its growth. The veins leading from the enlarged gland become distended, and in many cases those of the head and neck are subsequently dilated. Pulsation

FIG. 60.

A goître which caused dyspnœa by compressing the trachea between its lateral lobes.

in the carotids is occasionally increased, but in some instances the growth causes pressure on these vessels, and, by interfering with the blood-current, induces cerebral anæmia with spasm or convulsions. Spasm of the glottis or paralysis of the laryngeal muscles may also result from irritation of the recurrent laryngeal nerve. The most important result of enlargement of the thyroid is, however, interference with the passage of air through the trachea, which is usually compressed on either side by the two lateral lobes, but may in rare instances be narrowed by the pressure

exercised by the enlarged isthmus in front. In other cases the growth extends between the sternum and trachea, and squeezes the latter against the spine. Even in cases where no urgent dyspnœa exists, it is common for a bronchocele to produce some shortness of breath on exertion. The large majority of goîtres do not cause death, and a fatal termination is much more common in the rapidly growing tumours than in those of slower growth, even although the latter attain a greater size. The name **acute bronchocele** has been given to these quickly developing goîtres, and the dyspnœa which they cause is explained by the fact that the fasciæ and muscles of the neck have not time to stretch and provide room for their development; hence, all hollow and compressible structures necessarily suffer. These acute bronchoceles are most often met with in young subjects, and, in some instances at least, their growth is coincident with the changes that occur at puberty. In cases of cystic bronchocele, the cysts sometimes rapidly increase in size on account of the effusion of blood into their cavities, and may thus cause dyspnœa.

MALIGNANT DISEASE.

Sarcoma is rarely seen in the thyroid gland, but carcinoma is not uncommon. The latter growth usually affects thyroids which have previously been the seat of goîtrous enlargement, and is commonly diffused throughout the entire gland. The tumours are usually of the encephaloid variety, but occasionally they are scirrhous in type; sometimes they very closely simulate the normal glandular structure, and can only be distinguished from cases of simple hypertrophy by the infiltration of the neighbouring tissues and by their malignant clinical course. All growths of this class are liable to cause glandular enlargement, and to produce secondary growths in the viscera and bones. They usually run a rapid course, and terminate fatally, either by local complications or visceral disease.

Cachexia strumipriva is a term used to indicate a

peculiar condition allied to myxœdema, which is liable to ensue in patients whose thyroid glands have been excised.

Examples of this affection were first described by Kocher and Reverdin in the year 1883, and since then the observations of these surgeons have been confirmed by other operators. The disease commences insidiously from one month to four or five months after the operation, and is characterised by sensations of weakness, with dragging pains in the arms, or in all the extremities. Pains in the neck and trunk are less common. The pain is succeeded by a sense of chilliness, and chilblains are liable to form. The cerebral functions are early affected, and slowness of thought and of speech are especially noticeable. This condition is followed by further impairment of the mental capacities, with loss of memory and inability to acquire further knowledge, whilst gradually the patient passes into a condition of lethargy and feebleness. Coincident with the onset of the disease, swelling of the face, and especially of the eyelids, is noticed, and gradually, as the swelling spreads, the whole face becomes broad, the lips thick and pouting, the features lose all expression, and a semi-idiotic appearance results. The abdomen becomes large and tumid, the hands and feet are swollen and thickened, the skin as a whole becomes thickened and dry, and the surface epithelium readily scales in many cases. The hair becomes brittle, and commonly falls out in large quantities. Combined with these conditions there is always very marked anæmia, and in those patients who are children growth is almost entirely arrested, and the period of puberty is delayed.

It is now commonly believed that the foregoing symptoms are the direct result of removal of the entire thyroid gland, and the theories that they are due to injury of the sympathetic nerve, to atrophy or narrowing of the trachea, or to residence in a goîtrous district, are no longer credited.

But, although these typical symptoms may ensue in

many cases of thyroidectomy, they do not occur in all, and in others, although present, they are much modified. It is believed by some authorities that in all cases where they do not ensue, either the entire gland has not been removed, or that the accessory thyroid glands, which are known to exist in some people, subsequently hypertrophy, and take on the functions of the removed organ. It is indeed true that enlargement of accessory thyroids has been seen in many cases where the symptoms of cachexia strumipriva have not followed on thyroidectomy, but it is nevertheless probable that in some cases where no thyroid tissue at all is left, the typical symptoms of the disease in question do not supervene. In yet other cases the anæmia and the impairment of the cerebral functions occur without the swelling of the face and the changes in the skin, which are their usual accompaniments.

ACROMEGALY.

Acromegaly * is a disease which is characterised by great enlargement of the hands and feet, with much deformity of the face. The hands and feet are simply overgrown, all the tissues sharing in the enlargement, although there is in addition a great increase of fibrous tissue. The size of the extremities is indeed in advanced cases most remarkable, and the measurements of a hand, the cast of which is in the museum of St. Bartholomew's Hospital, compare as follows with the measurements of an average adult male hand:—

Length	9 in.	Normal hand,	$7\frac{1}{2}$ in.
Length of middle digit .	$3\frac{5}{8}$ in.	,,	$3\frac{1}{4}$ in.
Circumference at metacarpal bones . .	13 in.	,,	$8\frac{1}{2}$ in.
Circumference of middle finger	$4\frac{5}{8}$ in.	,,	$2\frac{3}{4}$ in.

The legs and forearms are not usually enlarged, but in some of the recorded cases the patients were very muscular. The spine usually presents a posterior curve in the dorsal

* ακρον, extremity; μεγαλη, large.

region, and there may be some lumbar lordosis or lateral curvature. The thorax is flattened from side to side and moves but little in respiration, the respiratory movements being chiefly abdominal. The skin is thick and often warty, and pendulous parts are specially thickened; the labia majora and the clitoris may be greatly hypertrophied. The growth

FIG. 61.

A hand from a case of Acromegaly and a normal hand.

of hair may be increased, and the hair itself may become very long and coarse.

The whole head is often enlarged, and the cranium tends to become lengthened antero-posteriorly, but the bones of the face are specially increased in size, and the jaw-bones more than the rest; the lower jaw by its great enlargement frequently projects in front of the upper teeth, and causes much disfigurement. The supra-ciliary ridges and the tarsal cartilages are much hypertrophied, and the whole nose is greatly increased, its bones, cartilages, and skin, all

undergoing overgrowth. The ears are not usually so much affected. On account of the alteration in shape of the maxillæ, complete closure of the teeth may be impossible. The lips are thick, and the lower lip tends to protrude and hang down; the expression of the features may be completely altered.

The youngest patient in whom acromegaly has been observed was fifteen years of age, and the oldest was sixty; but most of the cases have been observed between the ages of twenty and forty. Both males and females are attacked. Headache is almost always complained of, and there is often pain in the eyeballs. The general condition is one of lassitude, and the speech is usually thick and slow. The tongue is large and flabby, and appears too big for the mouth; the cartilages of the larynx in male patients are sometimes enlarged and thickened.

In addition to the changes in the extremities and face, blindness, with atrophy of the optic disc, loss of sense of smell and of taste, and cessation of the catamenia in women, have been commonly observed. Indeed, in all cases occurring in females the absence of normal menstruation is a constant phenomenon. Muscular weakness and excessive perspiration have also been observed in connection with acromegaly.

In patients on whom post-mortem examinations have been made, it has been found that the bones of the hands and feet were enlarged, that on all the bones there was a tendency to the exaggeration of normal ridges or tubercles, and that on some of the bones of the extremities, as well as on those of the face, there were osteophytic growths, although the general overgrowth of the whole bone was not seen in the extremities. The sternum is usually thickened, widened, and lengthened, and the ribs and clavicles are also greatly thickened; similar alterations are common in the pelvic bones. The uterus is generally atrophied and the vagina capacious.

The pathology of acromegaly is at present obscure, but

there is a tendency to attribute it to some disease of the pituitary body, the thyroid gland, or of the sympathetic nerves. It becomes more probable year by year that disease of the pituitary body is a constant factor in acromegaly, for in all recent post-mortem examinations this body has been found either greatly hypertrophied, the seat of new growth, or else completely atrophied. In a certain proportion of cases, the thyroid gland has also been found diseased. The diagnosis of acromegaly is generally easy. From the thick, heavy lips and broad, deformed face, the disease has been mistaken for myxœdema, but from the latter it differs very materially in the absence of dryness of the skin and hair of the extremities, and in the absence of any alteration in the mental condition, as well as in the positive deformity of the jaws, thorax, hands and feet. From osteitis deformans it may readily be separated, when it is remembered that in this affection the bones of the skull and the long bones are especially diseased, whilst in acromegaly it is just these bones that remain most unaffected.

CHAPTER XXXIV.

INJURIES AND DISEASES OF MUSCLES, TENDONS, AND BURSÆ.

Rupture of muscle.—Considering the constant strains to which they are subjected, muscles are but seldom torn, and, when they are ruptured, it is rather by a sudden and unexpected strain than by the employment of great muscular force. The ends of a ruptured muscle immediately retract, and remain separated by an interval which varies in extent in different cases. A certain amount of blood is always extravasated, and may form a considerable hæmatoma.

Union of the torn muscle is accomplished by a process of plastic inflammation, with exudation of lymph, and its subsequent organisation into fibrous tissue.

Wounds of muscle are followed by separation of the cut fibres, and in such injuries, as in rupture, repair is effected by fibrous tissue. Muscle may be said never to be reproduced, for, although attempts at reproduction of muscle-fibres have been described, the amount of new muscle thus formed is quite microscopic.

Inflammation of muscle.—Muscles are but little prone to inflammation, and when affected by so-called **myositis,** the latter is commonly the result either of injury, of inflammation spreading from surrounding parts, or of such constitutional conditions as gonorrhœal rheumatism, pyæmia and typhoid fever. The course of the inflammatory process in muscle does not differ from that in other tissues, and requires no special description.

Atrophy and degeneration of muscle.—The most common cause of muscular atrophy is disuse, and in cases where a limb has remained unused for years, as in chronic disease of a joint, the whole of the muscles may be so atrophied that no contractile tissue remains, the muscle-sheath being filled with a mass of fatty and fibrous tissue. The wasting of muscle which follows nerve-section has already been mentioned, and the various forms of atrophy and degeneration which are dependent upon disease of the central nervous system—*e.g.*, infantile paralysis, progressive muscular atrophy, &c.—do not come within the scope of the present work.

Ossification of muscle.—The formation of bony plates in muscle is a condition far more often met with in men than in women. Occurring in single muscles, it is by no means very rare, and is found most frequently in the deltoid and the adductors. The bony deposit occurs at the osseous insertion of the muscle, and appears to be produced either by constant strain or by frequent though slight contusions. The development of bone in the adductor muscles is most common in men who ride, and results in the production of the so-called "riders' bone."

In other, and fortunately rare, instances, the tendency to the formation of bone is found in many muscles, and cases are described in which the back and neck have thus become encased in a broad osseous cuirass, resulting in the complete immobility of the affected parts. The cause of such a condition is quite unknown. It has been named "**myositis ossificans**," and appears to be either of congenital origin or else to commence very soon after birth.

Tumours of muscle.—Primary tumours of muscle are rarely met with. The few cases that are recorded are almost all instances of sarcomatous growths. In four cases with which I am acquainted, the tumours were of rapid growth and ran a malignant course. The cells of which they were composed were mostly oval.

Syphilitic affections of muscles.—Gummata occur with tolerable frequency in the muscles, and appear to have a decided preference for those of the neck, and especially for the sternoid-mastoid. They often attain a considerable size, and may be as large as a hen's egg. They do not differ structurally from gummata elsewhere.

The chief **parasitic** diseases of muscle are those due to the presence of hydatids or of trichinæ. The latter are met with in voluntary muscles only.

INJURIES AND DISEASES OF TENDONS AND THEIR SHEATHS.

Wounds and ruptures.—Tendons are more liable to rupture than are muscles, notwithstanding their tougher and denser structure. The injury is at once followed by retraction of that portion to which the muscle is attached, whilst the other end does not alter its position except as the result of the movements of the part into which it is inserted. Repair is effected by a process of plastic inflammation. The exuded inflammatory products collect chiefly in the tendon-sheath, and in it undergo development, first into fibrous tissue, and subsequently into tendon. Repair is at once both rapid and complete, the newly formed tendon being capable of supporting considerable strain within a few weeks, and finally becoming indistinguishable, even by microscopic examination, from the neighbouring tendinous structure.

In wounds of tendons unaccompanied by the formation of pus—*e.g.*, in tenotomy for talipes—the process of repair is exactly the same as that above described; but in open wounds accompanied by much suppuration the tendons and their sheaths are liable to become matted to one another and to the surrounding parts in such a way that their future utility is either destroyed or greatly impaired. The prospect of union in such cases is greatly enhanced by careful suturing at the time of the accident, or even at a later date if repair has failed.

There are but few **diseases** to which tendons are liable, for they may be said to be never affected by inflammation or new growth except when secondarily implicated by extension from the surrounding parts. Gummata are rarely met with, but gouty deposits of urate of soda are not uncommon in the tendons of the feet and ankle. Fibrous tumours occasionally grow in connection with the tendon-sheaths of the fingers.

Teno-synovitis.—Teno-synovitis, or inflammation of the synovial lining of a tendon-sheath, is usually a subacute affection, and is most often seen in the extensor tendons of the wrist. The exciting cause is almost invariably excessive use of the muscles, and the constant friction of the tendons within their sheaths gives rise to roughening by the deposit of lymph, and a consequent creaking or crepitating sensation when the hand is placed over the affected part. The inflammation is usually transitory, and readily subsides with rest, but it is liable to recur when the muscles are again called upon for any unusual exertion.

In another class of cases the inflammation of the tendon-sheath is of **tubercular** nature. I have seen several such cases in which the tendons of the wrist or ankle were implicated, and in cases of tubercular arthritis it is very common to find an extension of tubercle to the tendon-sheath, the lining of which becomes as thick and pulpy as the synovial membrane of the joint.

Ganglion.—Ganglia are of two kinds—simple and compound. Each variety is most frequently seen in the sheaths of the tendons on either the palmar or dorsal surface of the wrist, or on the dorsal surface of the ankle. Ganglia are most common in those who are in the habit of using to excess the muscles of the wrist.

A simple ganglion is usually described as being a hernial protrusion of a portion of synovial membrane through an aperture in the sheath of the tendon, and it is supposed that the communication with the tendon-sheath being subsequently cut off, a small pouch of synovial

membrane is thus left outside it. It is much more probable that Paget's description is correct, which attributes the formation of a simple ganglion to a cystic degeneration of one of the synovial fringes normally present inside the tendon-sheath. If this be true, it follows that the fluid contained in the ganglion is never at any time in communication with the cavity of the synovial sheath, but is shut off in a cyst, which from the first is a closed cavity. However formed, a simple ganglion presents itself as a rounded, tense cyst, generally about the size of a hazel-nut, but occasionally larger, and containing a perfectly clear material of the consistency and appearance of glycerine jelly. The presence of a simple ganglion is liable to cause stiffness and pain in the affected tendon.

Compound ganglia are most common on the sheaths of the flexor and extensor tendons of the wrist. They are formed by a distension of the synovial sheaths with fluid, and may attain considerable size. In addition to sticky and inspissated synovia, they often contain numerous small, oval, or rounded, smooth masses of fibrin, of the size and appearance of melon-seeds. A compound ganglion always materially interferes with the movements of the tendons, and in time may completely cripple the hand.

Dupuytren's contraction is the name applied to a contraction of the fingers which results in some cases from injury or the use of some tool or instrument which causes pressure on, and irritation of, the tissues in the palm of the hand. In other cases it occurs in connection with the rheumatic or gouty diathesis. The little finger is usually first affected, and after it the ring. Dissection shows that this deformity is not caused by contraction of tendons, but by thickening of bands of fascia which are inserted into the base of the phalanges, and in time become adherent to, and cause puckering of, the skin. In cases of long standing the metacarpo-phalangeal and the first inter-phalangeal joints become more or less fixed, their articulating surfaces being in bad cases dislocated.

DISEASES OF BURSÆ.

The commonest disease of a bursa is simple distension with serous fluid. This distension is liable to follow chronic irritation of any kind, and, when the cause has been removed, will usually subside spontaneously.

If the irritation be kept up, chronic inflammation, with thickening of the bursal walls by fibrous tissue will ensue, and is occasionly combined with the formation of "melon-seed bodies," such as those already mentioned as occurring in compound ganglia. In some cases the walls of a bursa become so greatly thickened that the cavity is almost completely obliterated, and the bursa is practically transformed into a fibrous tumour.

Acute inflammation and suppuration of bursæ are also of common occurrence, and, if the bursal wall be not already thickened by old inflammation, the pus is liable to burst its way out, and to become diffused into the surrounding tissues.

Tumours of bursæ are very rare, but syphilitic gummata are common.

The bursæ which are most commonly the seat of all forms of disease are those over the ligamentum patellæ and the olecranon. Simple distension is also common in those situated over the tuber-ischii and the great trochanter, as well as in the bursa which lies in the popliteal space between the inner head of the gastrocnemius and the semi-membranosus.

CHAPTER XXXV.

DISEASES OF BONE.

ATROPHY.

Atrophy of bone, like the same process in other parts of the body, is the result of deficient use or of continuous pressure. The extreme wasting of the bones of stumps or of limbs with diseased joints affords an excellent example of the first cause, whilst the hollowing out and absorption of the sternum and vertebræ from the pressure of an aortic aneurysm well illustrate the second.

Two kinds of atrophy are described in long bones, but they frequently co-exist. **Eccentric** atrophy is a hollowing out of the bone from within, so that, whilst it maintains its natural shape and size when viewed from without, it is found, on section, to be reduced to a mere shell. Atrophy of this kind is often seen in aged people. In **concentric** atrophy the whole circumference and diameter of the bone are diminished, and its shaft is much more slender than is that of the opposite side. Such a bone, on section, is often found to be the seat of eccentric atrophy as well.

HYPERTROPHY.

True hypertrophy is not common. It chiefly results from increased blood-supply and from excessive use. Good examples of the former are occasionally supplied by cases of chronic inflammation in the neighbourhood of an epiphysis in a young subject, for, on account of the increased vascularity of the epiphysial bone, growth may be

stimulated to such an extent that, in the course of years, a limb may outgrow its fellow by several inches. Better examples of pure hypertrophy are afforded by cases where a bone is called upon to bear undue weight—*e.g.*, in congenital absence of the tibia, the fibula may be greatly enlarged; in hydrocephalus also the increased size of the brain is met by a corresponding increase in the cranium.

INFLAMMATION.

In considering the subject of inflammation of bone, it must be remembered that the osseous structure is practically ossified connective tissue, and that almost all that has been written in the chapter on "Inflammation" is strictly applicable to bone.

In osteitis, then, there are, first, hyperæmia and stasis, followed in quick succession by formation of new blood-vessels and exudation, with softening of the inflamed tissue, a change which plays a most important part in the course of inflammation of bone.

The blood-vessels of bone run in the cancellous spaces, the Haversian canals, and the canaliculi, and consequently it is into these pre-existing spaces that the exudation is poured. As in all inflammation, this exudation, or lymph, consists of cells and fibrin, and, as elsewhere, the lymph is subsequently vascularised, and forms what has already been called "interstitial granulation tissue" or "inflammatory new formation." The next step in most cases of osteitis is **rarefaction**, or thinning of the osseous structure. This is not, as was formerly supposed, the result of a mechanical expansion or dilatation of the bone-spaces by the mere pressure of the contained exudation, but is the result of the absorption and destruction of the bone itself by the exuded leucocytes which lie in contact with it. The walls of the canaliculi, Haversian canals, and cancellous spaces are eaten away by the cells, so that their naturally smooth contour becomes pitted and irregular; in this process of absorption the leucocytes are aided by

large giant cells, or osteoclasts, which are probably themselves derived from the connective-tissue cells. As the eroding process goes on, the bony substance becomes thinned away, or rarefied, and the cancellous spaces become larger, not only on account of the greater tenuity of their walls, but also because, where the osteitis is most advanced, several spaces are thrown into one by the complete removal of their boundaries. (See Fig. 62.)

FIG. 62.

Section of an Ilium, showing Rarefaction. The laminæ of the compact bone appear to be separated from one another, the cancellous spaces are enlarged, and in the lower part of the specimen an irregular cavity has been formed by destruction of the bone which formerly bounded the cancellous spaces.

A section of a bone in this stage of osteitis therefore reveals greatly increased vascularity, rarefaction of the osseous tissue, complete filling up of the spaces in the bone by red gelatinous masses of granulations, and softening of the bone itself, so that it may often be readily cut with a knife or broken down with the finger-nail. The compact-bone is no longer so dense, and its laminæ appear to be separated from each other. The cancellous spaces are so greatly enlarged that irregular cavities filled with soft pulp are formed, and the whole bone may be reduced to a mere shell. If the surface of a bone be the seat of osteitis, the smooth, compact tissue will become porous, rough, and pitted from erosion by the leucocytes, and the periosteum will be partly separated by the inflammatory exudation.

The further progress of a case of osteitis depends much on its cause and on the general health of the patient. Supposing that the cause has been an injury, and that the patient is healthy, the rarefaction will seldom or never proceed to such an extent as that described above, and, if the injury has been but slight, **resolution** and absorption

of the exudation will occur. If the osteitis has progressed further, organisation of the inflammatory products, with resulting **sclerosis**, is the most usual termination.

This process of sclerosis is precisely analogous to that of scarring in the soft parts. The cells of the granulations which occupy the cancellous spaces, Haversian canals, &c., become developed into connective tissue and finally into bone, the newly formed blood-vessels shrink and disappear, and the whole of the previously rarefied bone is converted into dense osseous structure, with fewer and smaller cancellous spaces than were present before the inflammation commenced. The inflammatory exudation which has collected beneath the periosteum also ossifies, and the bone is thickened by the formation around it of a layer of new bone formed for the most part in stalactitic or needle-like projections—an arrangement which results from the exudation collecting around the blood-vessels as they pass from the periosteum to the shaft.

Instead of terminating in sclerosis, osteitis may result in **suppuration**. This may occur in even the most healthy subjects, and is most frequent when the inflamed bone is exposed and thus rendered liable to septic infection. It may, however, result from want of rest, the presence of some irritating foreign body, the extension of inflammation from other parts, or from the bad state of health of the patient.

Whatever the cause, the disintegration of the bone by the interstitial granulation tissue progresses to such an extent that, finally, instead of the cancellous spaces being occupied merely by granulations, they are filled with pus. Similar changes occur on the surface, and pus is discharged from a granulating surface of inflamed bone, just as it might be from an ulcer in the soft parts. The pus having been discharged, and the cause of the inflammation removed, suppuration will cease, unless some constitutional condition causes it to continue. Lastly, organisation, with resulting sclerosis, will terminate the process, just as scarring

and contraction of the cicatrix terminate the process of ulceration in the soft tissues. In many cases of osteitis, suppuration and sclerosis co-exist; thus, in deep ulcers of the leg the tibia may be exposed and suppurating at one part, whilst the bone around is sclerosed and thickened.

It is to suppurative osteitis, with destruction of bone, such as is above described, that the term **caries** is often applied. Caries, however, is now rather a clinical than a pathological expression, and is used to signify inflammation of bone with rarefaction alone, as well as with suppuration, whilst by some authors it is limited to that form of osteitis which is called strumous. I myself think that, whilst the term caries is useful from a clinical point of view as expressing all forms of osteitis with destruction of bone, it is not advisable to employ it in a pathological work, on account of the various meanings which it has for both teachers and students.

FIG. 63.

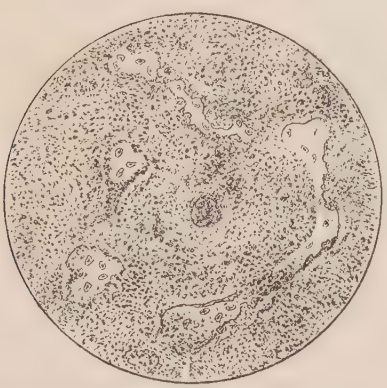
Section of Portions of the Tibia and Femur in a case of "Diffuse Tubercular Infiltration." The cancellous spaces are enlarged, and are in part filled by opaque masses of caseous matter.

In **tubercular** or **strumous osteitis**, as in other similar affections, there is a special tendency to caseation of the inflammatory products, as well as to a low and chronic form of suppuration; there is also little or no tendency to repair, or to arrest of the inflammation.

In such cases the inflammatory exudation in the cancellous spaces becomes converted into a soft pulpy mass of caseous matter and pus, and in the most typical examples there is no sign whatever of the formation of new bone, no appearance of sclerosis, none of repair. In this way, bones affected with tubercular osteitis may be hollowed out into mere shells—a condition which is best exemplified in the small bones of the carpus and tarsus. In some cases

minute portions of the osseous tissue necrose, and small sequestra of soft and crumbling bone are discharged with the pus. The name of "caries necrotica" has been applied to this condition. Three chief varieties of tubercular osteitis have been described: (1) The "circumscribed nodule," varying in size from that of a split pea to that of a bean, with a caseous centre and a red, gelatinous periphery. (2) The "diffuse infiltration," which is well shown in Fig. 63, and is characterised by a yellow caseous infiltration of large areas

FIG. 64.



Section of a Bone from a case of Tubercular Osteitis. The osseous tissue has been in great part destroyed, and its place taken by leucocytes. The remains of bony trabeculæ in process of destruction may be seen, and in the centre of the section is a caseous mass formed around a giant cell. (Crouch: $\frac{1}{6}$ inch.)

of cancellous tissue. (3) "Tubercular necrosis," which is almost limited to articular bone, and is described more fully in the chapter on "Diseases of the Joints."

If a bone in a state of so-called strumous osteitis be examined microscopically, it will be found that the process is essentially tubercular, and that tubercle and tubercle bacilli are mingled with the inflammatory exudation which fills the osseous framework. In most cases the bacilli are very few in number and difficult to demonstrate, though inoculation

with the tissue in which they lie will cause tubercle in susceptible animals, and will be followed by the growth of the bacilli in larger numbers.

Tubercular osteitis is most common in cancellous bone, and is seen especially in the articular ends of the long bones, in the carpus, tarsus, and bodies of the vertebræ. It is also one of the most common causes of strumous or tubercular disease of joints, for the process is essentially infective, and tends to spread to the tissues in the neighbourhood of the inflammatory focus.

A large proportion of cases of tubercular osteitis terminate in suppuration. The abscesses, at first chronic, are liable, after discharging their contents, to become septic, and to form pus in large quantities. Many patients consequently die of hectic fever, of amyloid disease, or of general tuberculosis.

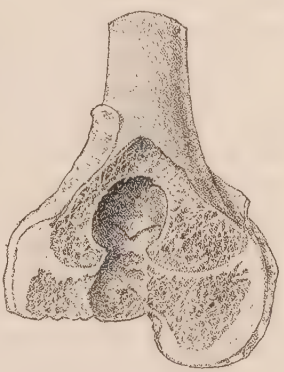
Abscess in bone.—Acute abscess never occurs in bone, for acute inflammations, which in the soft tissues would terminate in the formation of abscess, would in bone be sure to cause necrosis. Abscess in bone is, then, always chronic, and of slow formation.

The patients in whom these abscesses occur are always young, the most common age being between twelve and twenty. The bones most commonly affected are the tibia and the femur, and in these two bones, indeed, the larger number of all recorded abscesses will be found. Chronic abscess is never met with in the shafts, but always in the cancellous tissue of the epiphysial ends.

It is most probable that almost all these abscesses are of tubercular origin, like so many of the chronic abscesses in the soft tissues ; and this theory is in no way negatived by the clear history of injury which is occasionally given. Commencing as an osteitis of the cancellous tissue, rarefaction of the bone ensues, the walls of the cancellous spaces are destroyed, and thus a cavity is gradually formed in which the inflammatory products accumulate. As in the soft parts, the abscess-cavity soon becomes surrounded by newly formed

tissue, the result of a process of chronic inflammation, and thus the bone around the abscess becomes thickened and sclerosed. So long as the pus remains, the inflammation continues, and more and more new bone is produced from the superjacent periosteum. If, however, as is so often the case, the abscess is situated near to a joint, it is not shut in on this side, as elsewhere, by periosteal thickening, for there being no periosteum in this situation, new bone is never produced on an articular surface, and thus, if the abscess continues to extend, the pus is more liable to be discharged into the joint-cavity than elsewhere, and to cause suppurative arthritis.

FIG. 65.



The Lower End of a Humerus, showing an abscess which has burst into the elbow-joint.

In addition to the thickening of the bone itself, there is frequently thickening of the soft parts, and a gradual extension of the inflammatory process to the skin, with resulting œdema and reddening. Pain is tolerably constant, but, except at intervals, is not severe. There is generally a good deal of tenderness on pressure, and, on account of the proximity of the neighbouring articulation, attacks of synovitis, with effusion into the joint-cavity, are of frequent occurrence.

In some cases the formation of pus in the cancellous tissue is accompanied by necrosis of portions of the surrounding bone. Such cases, however, are usually much more acute than are those of simple abscess, and the danger of implication of a joint is much greater. Reference will again be made to these in the chapter on Diseases of the Joints under the heading of "Acute Epiphysitis."

Osteomyelitis.—Osteomyelitis is a term which is best limited to diffuse inflammation of the lining membrane and

the medulla of a bone, with implication of the surrounding cancellous tissue. Such a diffuse inflammation is always of infective origin, and is closely allied to cellulitis of the soft parts. It always originates in a wound which exposes the cancellous tissue or medulla, such as an amputation wound or a compound fracture. Osteomyelitis is characterised by the rapid formation of pus, which infiltrates the neighbouring cancellous bone, and is liable to extend along the whole length of the shaft. The inflammation extends not only in a longitudinal direction, but also through a varying thickness of the surrounding osseous structure, and may even reach the surface and affect the periosteum. An examination of a bone in a case of osteomyelitis reveals the following conditions :—The surrounding soft parts are inflamed, œdematous, and often sloughing. The periosteum is usually retracted from the bone in the neighbourhood of the wound, whilst from the medulla protrudes a soft mass of sloughing tissue. On section, the medullary canal is found filled with a similar material, composed of pus, broken-down medulla, and disintegrating bone. The cancellous spaces contain the products of inflammation in various stages of decomposition. The disease almost always results in necrosis, and the amount of dead bone is sometimes very extensive.

As has already been said, osteomyelitis is essentially an infective process, and, in consequence, there is often very considerable constitutional disturbance. Many patients die of pyæmia.

In some cases the inflammation becomes limited, and does not implicate the whole length of the shaft; and, indeed, it may be said that examples of diffuse osteomyelitis become more rare every day—a circumstance which is due to the improvements in the modern treatment of wounds.

DIFFUSE PERIOSTITIS.

The term "diffuse periostitis" is misleading, for the periosteum alone is never affected. In diffuse periostitis

there is a spreading, infective inflammation caused by pyogenic bacteria, and attacking both the periosteum and a varying thickness of the subjacent bone. The disease is essentially one of early life, and hardly ever occurs in adults.

The subjects of acute periostitis are often in an unhealthy condition at the time that they meet with the slight injury or the exposure to wet and cold which are the almost invariable causes of this affection. Occasionally, acute periostitis occurs during convalescence from one of the specific fevers.

As might be expected, the bones of the lower extremity, being the most exposed to cold and injury, are more frequently attacked than are those of other parts; the tibia and the lower and posterior part of the femur are most often involved. The humerus and clavicle seem to be more frequently attacked than the bones of the forearm.

It is probable that the local condition of certain parts of some of the bones further predisposes them to attack. Thus, the anterior surface of the tibia and that part of the femur which lies in the popliteal space owe their liability to acute periostitis to the fact that, on account of the absence of all muscular attachments, they are much less vascular and less protected than are those portions of the same bones which are closely covered by muscle. In some cases many bones are simultaneously affected.

The disease commences with local pain and swelling. The skin at first is not implicated, but soon becomes red and œdematous. Constitutional disturbance is generally marked, the temperature runs up to 103° or 104°, and rigors, vomiting, or convulsions may occur. The local swelling increases, and spreads along the bone, and in bad cases which are not subjected to treatment the inflammation may extend over the whole length of the shaft. Pyæmia is a very common complication, for the inflammatory exudation, being under considerable pressure, makes its way into the venous channels in the bone; and as these channels are

unable to collapse as they would in the soft tissues, the septic pus, mingled with blood-clot, is especially liable to mingle with the venous current and to be conveyed to the lungs.

An examination of the bone itself will reveal the following conditions :—The periosteum and the subjacent bone at first show the usual vascularity which marks the onset of inflammation. Very soon there is exudation, and pus is quickly formed. The pus infiltrates the looser and more cellular part of the periosteum, and extends from it into the soft tissues. It collects, however, in the greatest quantity between the periosteum and the bone, for here there is most room for it, and the periosteum is thus separated from the shaft to a variable extent. If the disease progresses, the stripping up of the periosteum may extend as far as the epiphysial cartilage. Beyond this it seldom extends, on account of the close attachment of the periosteum in this situation ; and, for the same reason, and on account of the difficulty the effusion has in making its way through the tough periosteum, the pus may extend between the epiphysial cartilage and the shaft, and may thus separate the one from the other.

FIG. 66.

A Tibia from a case of Acute Periostitis. The periosteum has been separated from the shaft and the greater part of the atter has necrosed.

Meanwhile, the inflammatory process also extends into the bone, and acute osteitis progresses as rapidly as does the periosteal inflammation.

The inflammation usually fails to reach the neighbouring joints, partly on account of the close attachment of the

periosteum at the epiphysis already mentioned, and also because the non-vascular epiphysial cartilage offers considerable resistance to the extension of the inflammatory process. Occasionally, however, the suppuration does involve the articulation, and sets up in it a most acute and destructive form of suppurative arthritis. If an exit be not provided for the pus, the latter will become diffused amongst the muscles, and will ultimately make its way through the skin by numerous apertures. If the pus be evacuated early, the disease may be cut short, and in favourable cases the bone recovers without necrosis. The extent to which the bone dies in any case depends not only on the separation of the periosteum, and the consequent interference with the blood-supply, but also, and probably to a far greater extent, on the inflammation of the bone itself. Acute osteitis is the most fertile cause of necrosis. and the greater the amount of bone inflamed the more extensive will be the necrosis. The separation of the dead bone from the living, and the formation of new bone, occur in the manner described below.

NECROSIS.

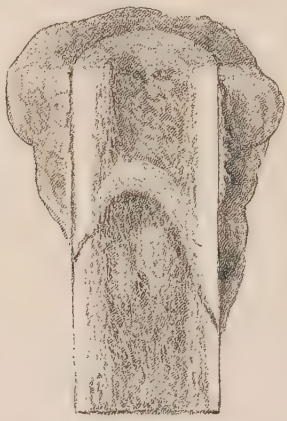
Necrosis of bone is the equivalent of gangrene of the soft parts, and, like the latter, it arises from interference with the blood supply.

Necrosis most frequently results from injury and acute inflammation. In some cases of the former the periosteum is torn away, and thus the bone is deprived of a considerable proportion of its blood. Such an injury is always followed by a certain amount of thrombosis in the torn blood-vessels, and, later on, by inflammation of the damaged tissues. Both of these tend to further interfere with the circulation.

In acute inflammation of any part of the body there is a tendency to the compression of the vessels by the inflammatory exudation. The more acute the inflammation the greater is this tendency, and the more dense and unyielding

the inflamed structure the greater is the tension of the extravasated fluid. In bone, the exudation is poured out into the cancellous spaces and the Haversian canals, and, in cases of acute osteitis, the blood-vessels are very quickly compressed by the pent-up exudation. In less acute cases, where the process of exudation is much slower, time is allowed for enlargement of the bony canals by absorption of their walls by the leucocytes, and thus more space is provided for the exuded fluid, and necrosis does not result. Again, necrosis is more common in compact than in cancel-

FIG. 67.



Portion of a Femur from a Stump, The lower inch of the bone is necrosed, the periosteum and the soft tissues are quite detached from it, and a line of demarcation has been formed between the dead and the living bone.

lous bone, for in the latter there is more room for the exudation, and consequently there is less tension.

The amount of bone which dies in any individual case depends chiefly on the extent of the osteitis, or, in cases of injury, on the extent of separation of the surrounding soft parts. In many instances these two causes are combined. Thus, in compound fractures the bone may be comminuted, and the fragments almost completely separated from the

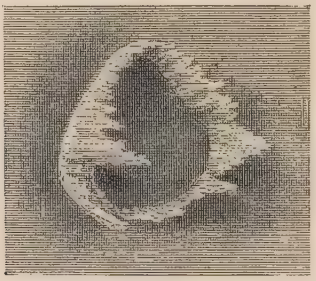
neighbouring tissues, whilst, on account of the extent of the injury, the resulting inflammation is proportionately great. When the periosteum is separated, and the superficial bone is alone injured, a thin shell of bone may alone die, whilst, in cases of diffuse periostitis and osteomyelitis, either the whole thickness or even the whole length of the shaft may perish. Necrosis of bone after amputations affords another example of the effect of injury. In these cases the inflammation which results from the sawing, and often from subsequent septic conditions, causes the death of a ring of bone which, though commonly very small, is in some cases an inch or more in depth. Under these circumstances, the stump refuses to heal, for the dead bone acts as a foreign body, and if an examination is made, the periosteum will be found detached from the necrosed extremity, which in time is separated and cast off. (Fig. 67.)

Separation of the dead bone.—When bone is dead, the living bone in contact with it shows signs of irritation. It becomes the seat of inflammatory changes, just as do the soft tissues in a case of gangrene; the inflammation progresses to ulceration, granulation tissue is formed, and the living bone is separated from the dead by a "line of demarcation." This process of separation is necessarily a slow one, and, where a considerable thickness of bone has to ulcerate through, many months may elapse before the process is complete. So long as the necrosed bone is in contact with the granulations of the neighbouring healthy bone, it is in part destroyed by the latter, and is eaten out and deeply pitted by the destructive action of the leucocytes. Consequently, if a piece of dead bone be examined, its under surface will be found rough and indented, and marked by numerous little pits or depressions which were once occupied by granulations. (See Fig. 68.) The surface of the dead bone is non-vascular and is usually white, but occasionally from exposure to the air, it becomes blackened.

The dead portion of bone when separated from the living is called a "sequestrum," and under favourable circum-

stances may be cast off from the body ; the ulcerated surface of bone left behind will then heal. If the necrosed bone is quite superficial, the process of shedding is called "exfoliation." This is most commonly seen in the bones of the skull, where destruction of the pericranium by mechanical injuries, or by burns, often results in death of the outer table alone, the inner table being supplied with blood from the dura mater.

FIG. 68.



A Ring of Necrosed Bone which has separated from a Stump.

Formation of new bone and inclusion of the sequestrum.—It has already been said that the separation of the dead from the living bone is the result of an inflammatory process. Now, this inflammation is not limited to the immediate neighbourhood of the necrosed bone, but extends over a considerable area. The living bone in contact with the sequestrum suppurates, but the osteitis set up around is less acute, and results in the formation of new bone. The latter is formed not only by interstitial deposit, but also, and probably to a greater extent, by the neighbouring periosteum and the surrounding soft parts. The amount of new bone formed may be very great, and it thus happens that, by the time the dead bone has become completely separated from the living, it is surrounded by a case of new bone of considerable thickness, and its extrusion or exfoliation is prevented.

This formation of new bone is often well seen in cases of acute periostitis, where, a considerable length of the shaft having been destroyed, the sequestrum becomes surrounded by a sheath of new and porous bone, except at one or two places where discharging sinuses open through apertures in the new bone on to the skin surface of the limb. These apertures in the bony sheath are called "cloacæ," and occur in places where the periosteum has been previously destroyed by sloughing. But, although such a replacement of dead bone by newly formed osseous structure is often most useful in restoring the functions of the part, it is not without its disadvantages. In the larger number of cases the sequestrum becomes so much shut in that its extrusion by natural means is impossible, whilst, so long as it remains, suppuration is kept up, chronic osteitis and periostitis persist, and the case of new bone becomes thicker and thicker. In cases where this thickening occurs at the articular end of a bone, it may be so extensive as ultimately to limit the movements of the joint, and the latter may become anchylosed by an extension to it of the inflammatory changes in progress in the parts around. It is evident, therefore, that it is advisable to remove the sequestrum as soon as it has become sufficiently loose; but it is not well to anticipate matters by artificially separating the dead bone from the living, for the reasons that, until the line of demarcation appears, it is not possible to be sure of the extent of the necrosis, and that, if the dead bone be

FIG. 69.

A Tibia, showing necrosis of the shaft and formation of periosteal new bone.

removed, the formative periostitis will soon cease, and the production of new bone will be arrested. Where large portions of the shaft have been destroyed, such a failure to form new bone may result in the permanent impairment of the functions of the limb.

Perhaps the most troublesome cases of all are those of central necrosis—cases which depend apparently on a limited osteomyelitis—for in these the bone which dies is that which bounds the medullary canal, and from the beginning it is shut in by the subjacent compact bone.

Necrosis without suppuration.—Mr. Morrant Baker has drawn attention to the occasional occurrence of necrosis without external suppuration, and the subject is one of so much importance that it is well worthy of consideration.

In cases of this kind a history of injury may or may not be obtained, and the attention of the patient is attracted by a swelling on a limb, which increases in size with some rapidity, and as often as not is painless, and not even tender when examined. The superjacent skin may be reddened, but it is sometimes quite natural. The swelling feels firm and often elastic. The accompanying drawing of a femur is from a case of this kind. The patient was a lad of nineteen, who for about three months had noticed such a swelling as I have above described; it was painless, and increasing in size. The case was diagnosed as either one of periosteal sarcoma or of necrosis without suppuration, and it was decided to make an exploratory incision to settle the question. This accordingly was done, and a mass of soft gelatinous tissue was exposed, which, in the opinion of these present, was of a sarcomatous nature. Amputation was therefore performed. A section of the bone after removal showed that a very small portion of the compact tissue of the shaft had necrosed, and that it had been subsequently shut in by a quantity of new bone formed around. Outside this new bone was a mass of fibrous tissue and infiltrated muscle, the section of which had simulated that of a sarcoma. The size of the swelling was

altogether out of proportion to the amount of the necrosis; about twenty or thirty drops of pus lay around the sequestrum, and communicated by a minute aperature in the new bone with a tiny cavity in the inflammatory tissue.

This case well illustrates the chief difficulty in the diagnosis of this form of necrosis, for, although its true nature was suspected, even a free incision rather obscured than cleared it up.

FIG. 70.

Section of a Femur, showing excessive formation of new bone, and thickening of the soft tissues around a small sequestrum.

In addition to those forms of necrosis which result from injury, or from inflammation started by either mechanical or septic agents, it is necessary to allude to the necrosis which results from **chemical poisons**. Of these there are but two whose influence is of importance—mercury and phosphorus. They act differently, for the former causes necrosis through its absorption into the system, whilst phosphorus acts locally upon the jawbones through the inhalation of its fumes with the respired air. The effect of mercury on the teeth and gums is well known, and its implication of the jawbone is but an extension of the same influence to deeper structures. With very rare exceptions, phosphorus, when taken internally, does not cause necrosis, and its fumes may also be safely inhaled if the teeth are in a sound state. If, however, there is dental caries, a low form of persistently spreading osteitis and periostitis is started which often results in necrosis of considerable portions of the maxillæ. The separation of the sequestrum and the formation of new bone do not differ from similar processes in other parts.

CHAPTER XXXVI.

DISEASES OF BONE—(*continued*).

SYPHILITIC DISEASES OF BONE.

Disease of the bones is one of the commonest manifestations of constitutional syphilis, and may occur either early or late in the course of this affection. In the early stage of secondary syphilis, pains in the bones, which are worse at night, are of frequent occurrence, and, although actual disease is not always to be discovered in a patient with these symptoms, the formation of nodes is very common.

A node is a localised inflammatory swelling on a bone. The commonest situations of such swellings are the long bones, especially the tibiæ, and the bones of the skull. A section of a node exhibits inflammatory exudation into and beneath the periosteum, as well as into the surface layers of the bone itself. In a large number of instances much of this exudation is subsequently absorbed, and the bone is restored to its natural condition; but in other cases, especially if proper treatment be not adopted, the exudation undergoes organisation, first into fibrous tissue, and subsequently into bone, and a so-called "hard node" is formed. The new bone is generally rough and porous, like new bone elsewhere.

The tendency to organisation, however, is dependent both upon the constitution of the patient and also on the locality of the node. If the patient be otherwise healthy, and the node be on a long bone, organisation is likely to ensue, but if the patient be broken down in health, and especially if

the skull be the seat of inflammation, suppuration is liable to follow, and a "soft node" is produced. In this case a small periosteal abscess is formed, which, when opened, is found to be connected with a carious condition of the subjacent bone, and indicates that there has been a localised suppurative periostitis and osteitis, with rarefaction and partial destruction of the superficial layers of bone.

But whilst nodes are of common occurrence, they can scarcely be reckoned as serious lesions, when we consider how much more severely the bones may suffer in syphilis. For, instead of there being but one or two isolated nodes, the whole length of a bone may be attacked by chronic osteitis and periostitis, resulting in the formation of new bone, deformity of the limb, and great pain. In other cases, again, this sclerosis may be accompanied by caries and necrosis, and discharging abscesses and sinuses may tend still further to damage the health of the patient. The necrosis is chiefly caused by the cutting off of the blood-supply by the pressure of the inflammatory exudation, but is also attributed by some authors to the obstruction to the circulation which is caused by the sclerosis of the surrounding osseous structure. It is difficult to exaggerate the extent to which a bone may be damaged; caries, necrosis, sclerosis, and formation of stalactitic periosteal growths may so alter it as to render it almost unrecognisable.

It is, however, in the bones of the skull and face that syphilis is seen at its worst, for here the destructive processes far outrun those which cause mere sclerosis and osteophytic growth. Ulceration extending from the nasal or buccal mucous membrane may implicate the hard palate and the bones and cartilages of the nose. Much of the latter organ may be destroyed, and perforation of the palate may cause difficulty in swallowing, with tendency to the return of fluid through the nostrils, and nasal intonation of the voice. Inflammation of the middle ear may cause necrosis of the auditory ossicles and of the temporal bone, and thus produce deafness, or, by an extension of inflamma-

tion, may cause meningitis or suppuration in the brain itself.

The vault of the skull is perhaps the favourite seat of syphilitic inflammations. Here the disease may attack either the inner or the outer table, and in some cases implicates both. Caries and necrosis go hand in hand, portions of bone being first ulcerated, and then gradually cut off from their vascular supply by an extension of an ulceration.

FIG. 71.

Calvaria with extensive Syphilitic Ulceration. The frontal bone is perforated in several places.

Three varieties of syphilitic ulceration of the skull have been described—the annular, the tuberculated, and the reticulated. In the annular form the ulceration commences at one spot and spreads eccentrically from this as a focus. The bone becomes worm-eaten and pitted, a circular groove is then formed around it, and the central ulcerated portion is separated from its vascular connections, and dies. In the reticulated variety there is a network of periosteal new bone, which is subsequently destroyed by annular ulcers, such as those above described. In the tuberculated ulceration there is first the formation of raised, rounded, tubercular

nodules of new bone, and subsequently an ulceration and destruction of them.

These varieties of ulceration frequently co-exist in the same patient, whilst the amount of necrosed bone differs much in different cases. The meninges do not usually become inflamed, and the brain itself likewise escapes. Such, however, is not always the case, for death may ensue from suppurative meningitis, due to an extension of inflammation from the carious bone. (See also Congenital Syphilis.)

RHEUMATIC AFFECTIONS OF BONE.

The most commonly recognised form of rheumatic disease of bone is a chronic periostitis, with thickening, and the formation of so-called "rheumatic nodes." These nodes are usually situated upon the long bones, and are frequently indistinguishable from those due to syphilis. They never suppurate, however, and are sometimes more diffused than the syphilitic variety. The affections of the articular ends of the long bones in osteo-arthritis will be described in the chapters on Diseases of the Joints.

OSTEITIS DEFORMANS.

This is a form of very chronic inflammation of bone occurring in people past middle age, implicating many bones, and accompanied by a peculiar softening and bending of the osseous structure. It was first described by Sir James Paget.

The bones affected by this disease become gradually thickened by the deposit of new bone from the periosteum, and by the same process their normal outlines are slowly obliterated. By the exudation of inflammatory products within the bone the osseous structure is absorbed, rarefied, and softened, the spaces thus formed being filled with inflammatory exudation. The whole bone becomes greatly increased in circumference, and, on section, is seen to be

much thickened. In some cases the medullary canal is increased in size.

In consequence of the softening which accompanies the inflammatory process the bones become bent, the normal curves being at first increased, whilst, after a time, fresh curvatures are developed.

The skull is increased in thickness, the forehead becomes large and prominent, and the face in consequence appears to be too small for the cranium, by which it is overshadowed. The clavicles become much curved and thickened. The thorax falls in on account of the yielding of the softened ribs, and the abdomen becomes prominent. The femora curve chiefly outwards, and the tibiæ forwards. The humerus does not curve so much as most of the long bones, but the radius and ulna curve backwards. In consequence of the bending of the bones of the lower extremity, and of the general posterior curvature which is often met with in the spine, the height of the patient is frequently diminished by several inches. The walk is tottering, and the support of a stick is often necessary. The shoulders fall forward over the chest, and the head protrudes in a very peculiar manner, looking as though it was too heavy for the cervical vertebræ, for the chin would naturally rest upon the sternum, and, in order to look up, the patient thrusts it out so that the face is carried on a plane which is considerably anterior to that of the body. The course of the disease is slow, and usually extends over many years. A fatal termination may result from the difficulty of respiration caused by the softened thoracic wall.

FIG. 72.

Section of a Tibia from a case of Osteitis Deformans.

FIG. 73.

Figure of a woman with Osteitis Deformans, showing the curvature of the lower extremities, the large forehead, and the peculiar carriage of the head.

The disease has no known cause, and no remedial measures of any importance have been discovered. It seldom occurs before the age of forty, and is most common in males. It has been associated with gout in some patients, and with malignant tumours in others, but no causative relation with either of these has been established.

MOLLITIES OSSIUM.

Mollities ossium is a disease in which there is a gradual softening and subsequent bending of the bones. Many parts of the skeleton are usually involved at the same time, and the diseased condition affects the whole of the bones both of the limbs and of the trunk. Women of middle age are infinitely more subject to mollities than are men, and in a large number of cases it undoubtedly commences during pregnancy. In some patients mollities is limited to the pelvic bones. No time of life is altogether exempt, and examples have been recorded in infants as well as in extreme old age. In some few cases the course of the disease has been arrested either by nature or, apparently, by treatment.

An examination of the affected bones shows a gradual destruction of the cancellous tissue, with a corresponding increase in the medulla; the destruction extends to the compact bone, the osseous structure is gradually removed, the bone becomes soft and yielding, and, but little of its animal matrix remaining, it is in great part decalcified. In the process of destruction there is no sign of inflammation or of new growth: the bone salts appear to be simply dissolved out. An examination of the medulla shows, in some cases, an increase of fat, and in others a peculiar gelatinous matter which is chiefly composed of oil, fat, and disintegrated blood-cells. The osteoclasts which are always present during bone destruction from any cause are here also increased in number.

The true pathology of mollities is yet obscure. It has been attributed to an excess of lactic acid in the blood, but

this has certainly not been clearly demonstrated. When occurring during pregnancy, it has been attributed to the demand by the fœtus for bone salts; but this does not explain the cases which occur independently of pregnancy. In some cases the disease has been associated with the growth of malignant tumours in other parts of the body, and it has been suggested that mollities itself is nothing more than a diffuse tumour growth. Very little reflection is required to dispose of this theory. Lastly, mollities has been attributed to some influence of the nervous system, but what it is, or how it acts, is quite obscure.

RICKETS.

Rickets is a constitutional disease in which the bones are specially liable to suffer; they are not the only tissues diseased, but, on account of the very obvious changes that occur in their shape and structure, and of the importance of these changes in the further development of the body and limbs, the diseased state of the bones attracts attention rather than the constitutional cachexia and the visceral disease which accompany it.

Rickets is practically never congenital (the so-called cases of congenital rickets being properly referred to sporadic cretinism and "achondroplasia"), but is chiefly due to bad feeding, although any bad hygienic conditions, such as overcrowding, deficient ventilation, &c., undoubtedly influence its development. Under the head of bad feeding is to be included, not only deficient quantities of food, but the ingestion of food which is not readily assimilable, or is likely to set up gastric or intestinal catarrh; thus the feeding of young infants with starchy material, such as biscuits and bread, is extremely harmful.

Rickets is most commonly developed during the first two years of life, but may occur as late as seven or eight years of age, whilst isolated cases are recorded in patients of twelve or fourteen. Children in towns are more frequently affected than are those in the country, and for obvious

reasons. The general signs of rickets, independently of the osseous affections, are briefly as follows:—The child is liable to attacks of vomiting and of diarrhœa, its food evidently causes dyspepsia, and the abdomen becomes considerably swollen. There is much lassitude, with unwillingness for exertion and impairment of muscular power and tenderness of the limbs. At night the child sweats much, especially about the head, and often throws off the bedclothes. The teeth are late in being cut, and the anterior fontanelle remains open for an undue length of time. The liver, spleen, and lymphatic glands may become enlarged, and laryngismus stridulus or bronchitis may occur as complications.

As regards the bones, the following changes may be noticed:—The long bones become excessively curved, the bending being at first due to an exaggeration of the normal curves, although, later on, secondary curves are developed. The articular ends of the long bones become swollen at the line of junction of the diaphyses with the epiphysial cartilages, whilst similar swellings developing at the sternal extremities of the ribs produce the so called "beads" on the latter bones. On account of its softened state, the thoracic wall fails to resist the negative pressure established within the thorax during the act of inspiration, and its upper part becomes compressed against the yielding lungs; the lower portion of the thorax is not thus driven in, being supported by the solid liver and spleen and the other abdominal viscera. The spine becomes more curved than natural, and in some cases a general posterior curve takes the place of the normal sinuous outline. The head is enlarged, and the forehead especially is bulging and prominent.

The iliac crests are turned out and the pelvis is either compressed in an antero-posterior direction by the weight of the body being transmitted through the spine and the sacrum when the patient occupies a sitting posture, or else is flattened from side to side by the pressure of the heads of the femora when the patient is supported on the lower

extremities. Thus, the former deformity is more common when the disease affects children unable to walk ; the latter, when older patients are attacked.

Rachitic children are generally undersized, though they may be fat and flabby, and in bad cases the bone disease appears to act as a deterrent to future healthy growth, for a patient who has suffered severely from rickets seldom attains middle height. In consequence of the softened state of the bones, greenstick fractures are not uncommon.

An examination of the bones themselves after removal from the body shows that the osseous lesions above described are due to an imperfection in the calcification of the growing bone, and that, whilst the removal of old bone and the formation of the animal matrix for the new bone both proceed normally, it is in the failure of deposit of calcareous salts in this matrix that the real osseous defect in rickets exists. There is not, on the one hand, an increased absorption of bone, nor is there, on the other, an increased preparation for ossification.

In the normal increase of a bone in thickness the more central portions of the cancellous structure are gradually removed by absorption, so that the medullary canal slowly, but constantly, increases in size. And in a healthy bone, just as fast as this hollowing out progresses within, so fast is new bone laid down by the surrounding periosteum, the proper proportions of the compact bone and of the medullary canal being thus maintained. Now, in rickets, the former part of this process progresses quite naturally. The shaft is hollowed out from within in a perfectly normal manner. It is the formation of new bone from the periosteum that fails, and, as the old bone is gradually removed, layer on layer of the animal matrix of the new bone is certainly formed from the periosteum, but being only imperfectly calcified, the shaft comes to be gradually formed of soft unresisting bone, and consequently becomes curved and deformed. It is seldom that all attempts at calcification are entirely absent ; there is generally some attempt,

however slight, and the result of this is the formation of an imperfectly developed bone from the periosteum, to which the name of "osteoid tissue" has been given.

At the epiphysial ends changes of a precisely analogous nature are found; here also there is growth with imperfect calcification. The epiphysial cartilage in a normal bone is continually growing, but just as fast as it grows, so fast does calcification extend into it from the diaphysis, and thus the cartilage itself never exceeds certain limits of size.

FIG. 74.

Section through the Line of Ossification of a Rachitic Bone, showing the irregularity of the rows of cartilage cells and the imperfect calcification. (Zeiss, A.)

Now, in rickets, the growth of the cartilage progresses just as it does in a normal bone, but calcification is slow and imperfect. It thus happens that in a section of a rachitic bone the epiphysial cartilage is found to be considerably in excess of that which is natural. Yet here also, as in the case of the formation of new bone from the periosteum, calcification is not absent, but only incomplete; consequently, the line of ossification, instead of being regular and even, becomes irregular and jagged; islands of calcareous

material are found in the enlarged epiphysial cartilage, and portions of unaltered cartilage are left behind in the most recently formed new bone. Microscopical examination shows great irregularity in the rows of cartilage cells, and imperfectly formed bone. (See Fig. 74.)

The swellings at the junctions of the epiphyses with the

FIG. 75.

"Beads" on the inner surface of rachitic ribs.

shafts are now readily explained. The imperfectly formed new bone is softer than natural, and, being compressed between the epiphysis on the one hand and the diaphysis on the other, it bulges at the circumference, as would any other soft and yielding structure.

By most writers on the subject it is stated that in rickets

there is "increased preparation for the formation of new bone," *i.e.*, increased growth of the epiphysial cartilage, and the increased width of the latter in rickets is by such observers considered to be the result of the increased growth, and not, as is above described, of delayed calcification. The question is one which is a little difficult to settle definitely, but it is on the face of it highly improbable that a disease which results from malnutrition would cause increased growth of any tissue. As a matter of fact, also, the bones of such patients, instead of being longer, as they would be if the epiphyses grew abnormally fast, are shorter than natural, and it is well known that rickety children are usually ill-developed and stunted.

In exceptional cases the bones in rickets undergo further change, and becoming greatly increased in thickness, lose much of their natural shape. This, again, is considered to be the result of a tendency to increased growth, but should in my opinion, be looked upon rather as the result of the rickets than as an essential part of the morbid process. The thickening is probably of the nature of a compensative hypertrophy, and is designed to support the weak and yielding bone, whilst, on account of the rachitic condition of the patient, all the new osseous tissue thus formed is porous and spongy, as already described.

After rickets has ceased, the bones commonly become denser than natural, and growth is often arrested at an earlier age than usual. When the bone has been curved, it is especially thickened on the side of the concavity—a condition which evidently strengthens it and tends to prevent the curve from increasing.

Scurvy rickets.—This name has been given to a disease which, like rickets, arises from bad feeding, and may be associated with the latter affection. It is characterised by effusion of blood beneath the periosteum of one or many of the bones, together with hæmorrhagic extravasations in the soft tissues, spongy gums, and purpuric eruptions. The bones of the lower extremity are more frequently affected than are those of the upper, and the effusions of blood are

most common at the junctions of the diaphyses with the epiphysial cartilages: in some cases separation of an epiphysis occurs.

The swellings caused by the hæmorrhage are quite unsymmetrical, and vary much in size from time to time, the disinclination to use the limbs is much more noticeable than in rickets, and there is frequently much pain and tenderness. The disease is not uncommonly fatal, and an examination of the bones after death sometimes shows that the sub-periosteal hæmorrhage has been very extensive. Notwithstanding the separation of the periosteum, necrosis never ensues.*

FŒTAL RICKETS—SPORADIC CRETINISM—ACHONDROPLASIA.

Rickets is never a congenital disease, but the name of "fœtal rickets" has been introduced to indicate a condition of the skeleton which bears a superficial resemblance to true rickets. Infants and children the subjects of the disease in question are of stunted growth, with large heads, short and curved limbs, thick fat necks, and imperfect mental development. As they grow up, the face becomes broad and flat, the nose flat and thick, the lips heavy and pouting, and the expression dull and heavy. Puberty occurs very late, and the sexual organs often remain permanently undeveloped; a patient of five and-twenty or thirty frequently looks no more than eight or ten years of age, whilst his cerebral functions are usually still less developed than is his body. Many of these people are idiots. In some cases the thyroid gland is enlarged; in others it is absent; in others again, it appears to be normal. The supra-clavicular fossæ are often occupied by diffused fatty growths. On account of the resemblance of the patients who are the subject of this disease to the cretins of Switzerland and Italy, the name of "**sporadic cretinism**" has been applied to this class of cases.

* See a paper by Dr. Barlow in *Trans. Med. Chir. Soc.*, vol. lxvi.

An examination of the skeleton shows the following changes:—The base of the skull is often short, and the basi-occipital and basi-sphenoid bones may be prematurely united: the vault of the skull is fully developed, and there is sometimes hydrocephalus. The clavicles (which, like the vault of the skull, are formed in membrane) are of normal length, as is also the vertebral column. The diaphyses of the bones of the extremities are very short, thick, and

FIG. 76.

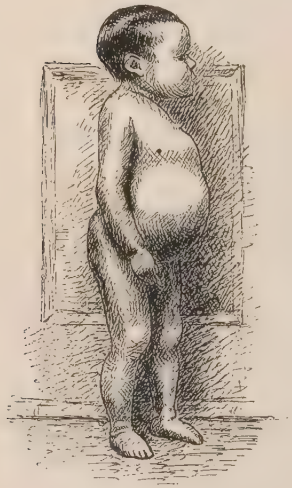


FIG. 77.

A sporadic cretin aged nine years.

curved: in some cases they are only represented by most minute portions of bone. The epiphyses are large and well formed. The sternal extremities of the ribs terminate in cup shaped cavities, which surround the ends of their corresponding cartilages.

Microscopical examination shows an ingrowth of fibrous tissue between the epiphysial cartilage and the diaphysis, combined with the almost complete absence of all attempts at ossification. The true pathology of sporadic cretinism is uncertain, though, reasoning by analogy, it appears probable

FIG. 78.

Photographs of two brothers affected with Achondroplasia.

that it is dependent upon some alteration in the functions of the thyroid body.

Achondroplasia.—This term has been suggested as a suitable designation for certain cases of deformity of the limbs which are evidently due to imperfect ossification of the epiphysial cartilage, and to a consequent shortening of the limbs. It has lately been pointed out by Parrot, Kaufmann, and Dr. John Thomson of Edinburgh, that cases of this class have been erroneously included amongst those of "fœtal rickets," and "sporadic cretinism," and it is certain that in the cases in question there is no real rickets and no disease or abnormality of the thyroid gland. It is further probable that many of the specimens described as fœtal rickets or sporadic cretinism really belong to this class, and have not hitherto been sufficiently recognised.

The abnormal condition commences in utero, and the bones which are chiefly affected are those which are the earliest to be developed, "and those which though formed in cartilage, remain altogether or mainly cartilaginous till a late period of intra-uterine life, are found quite normal in size" (Thomson), so that the morbid process must commence at a period when the differentiation of the mesoblast is in progress.

In the most marked examples of this affection the fœtus is generally still-born; very few survive birth more than a few days. In those cases where early death does not occur the patient grows up a dwarf, with curiously thickened and stunted limbs, but with a spinal column of normal length, and with normal clavicles, scapulæ, hands and feet, the deformity thus affecting mainly the limbs and pelvis, as in sporadic cretinism, and as in the latter disease, the base of the skull is imperfectly developed. In these patients, however, there are none of the associated mental conditions of cretinism; the skin and hair are natural and the sexual organs are normally developed. I am much indebted to Dr. Thomson for the accompanying illustration taken from his paper in the *Edinburgh Medical Journal*.

CHAPTER XXXVII.

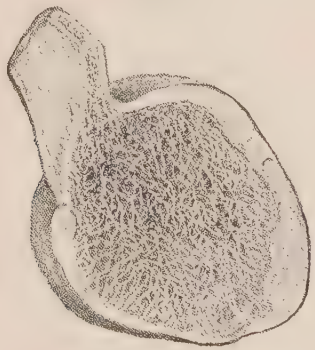
TUMOURS OF BONE.

EXOSTOSES.

Exostoses, or osteomata, are the most common tumours of bones. There are two chief varieties of such growths—**cancellous** and **ivory.**

Cancellous exostoses are composed of cancellous tissue exactly similar in structure to that which forms the

Fig. 79.



Section of a Cancellous Exostosis, covered with Cartilage, from the Femur.

articular ends of the long bones. They occur almost invariably in young people, and are most commonly seen on the ungual phalanx of the great toe or at the articular ends of the long bones, especially on the lower and inner side of the femur just by the adductor tubercle, and on the upper

end of the tibia. They appear to grow from portions of the epiphysial cartilage which have failed to become ossified, and which subsequently take on active and independent growth. A section of a growing cancellous exostosis will always show a covering of cartilage, and microscopical examination will reveal the formation of new bone in progress, just as does a section through an epiphysial cartilage.

Cancellous exostoses seldom attain any considerable size, being most commonly about as large as a walnut. They usually cease to grow at the time when the epiphysis is united to the shaft—a point which is worth remembering when considering the advisability of removal. They are often attached to the bone by a distinct pedicle, and, especially when they have been subjected to pressure or friction, may be covered by a bursa, which may render their hardness less evident than would otherwise be the case. Occasionally, these bursæ are in direct communication with the neighbouring articulation.

Cancellous exostoses are sometimes multiple, and as many as one hundred or more have been known to occur on a single individual. These multiple exostoses are frequently hereditary, and are often found in several members of the same family. In their structure and mode of growth, as well as in their preference for the articular ends of long bones, they do not differ from the single exostoses.

Ivory exostoses are composed of compact bone of more than usual density, with fewer Haversian spaces and canaliculi than are present in normal bone. Like the cancellous growths, they are most common in young people, but occur also in middle age. The most usual situation for such tumours is the cranium, but they are also found on the bones of the face and on the ilium and scapula. They are usually single and small, often not larger than a hazel-nut. They are in no way dangerous to life, but, from their situation, may be the source of much inconvenience, and, when growing from the bones of the orbit, may cause so much pressure on the eyeball as to seriously interfere with the

sight, or even to destroy the eyeball itself. So great is the density and hardness of the bone of which they are composed that in some cases all attempts at removal by bone forceps or saws have been frustrated.

The special forms of osteomata which grow in the jaws are described under "Tumours of the Jawbones."

ENCHONDROMA.

Cartilaginous tumours grow almost exclusively on the bones of the hand, and much more rarely on those of the foot and on the ribs. Pure enchondromata also occur at the articular ends of the long bones of the lower extremity, but most of the cartilaginous growths in these situations are mixed with sarcomatous elements, and should be classed amongst the malignant tumours.

Enchondromata of the hand are frequently multiple, and grow on the phalanges more often than on the metacarpal bones. In bad cases, the hand is completely disfigured and useless. The growth commences inside the affected bone, and expands the compact tissues so as to form a bony shell. Unless the tumour is of unusual size, it can be enucleated without sacrifice of the entire bone.

FIG. 80.

Enchondroma of Finger.

The cartilage of which these tumours is composed is almost always hyaline, but not infrequently the matrix shows in parts traces of fibrillation. Neither ossification nor calcification is frequent.

FIBROMA.

Fibrous tumours of bone are of limited distribution, and, indeed, appear to be almost confined to the nasal and buccal cavities. In the former they occur as fibrous polypi, and in the latter as fibrous epulides. They spring from the periosteum in all cases, and are of an innocent nature.

CYSTS.

Cysts containing serous fluid, such as are met with in the soft tissues, are not found in bone, whilst mucous cysts, and others which occur in the maxillæ, are dealt with under "Tumours of the Jawbones." The only other fluid tumours met with in the bones are of a parasitic nature, and contain hydatids. Cysts of this nature may attain a considerable size, expand the bone, and lead to its fracture. Their intimate structure does not differ from that of hydatids in other parts of the body. Secondary, or degeneration cysts, are common in the endosteal sarcomata.

SARCOMA.

Sarcoma is the only form of malignant tumour which occurs primarily in bone. Carcinoma cannot originate in osseous tissue, for its growth always commences in pre-existing epithelium.

Speaking generally, sarcoma of bone may be said to be a disease of early life, though not of infancy. By far the larger number of cases occur between the ages of fifteen and thirty, though examples are to be found in patients both older and younger than these limits.

Sarcomata are most conveniently divided into two main classes—those which grow outside the bone, or **sub-periosteal sarcomata**, and those which grow within, or **endosteal sarcomata.** The growth in either case may be composed of round, spindle, or mixed cells, whilst in the endosteal sarcomata, and very rarely in the periosteal ones, myeloid or giant cells are found. In all the varieties, chondrification, ossification, and calcification may occur, and thus, much of the growth may be composed of cartilage, bone, or calcareous material. Such an alteration in structure does not affect the clinical character of the tumour or modify its malignancy, but the secondary growths, if such be present, will closely simulate the primary tumour.

Some bones are much more frequently diseased than are others, those most commonly affected being the femur, tibia, humerus, the bones of the skull, and the lower jaw.

The **sub-periosteal sarcomata** originate in the periosteum, and separate it from the subjacent bone. As they increase in size, they extend through the periosteum and infiltrate the surrounding soft tissues, and, in addition, grow into the bone itself, and develop in the osseous structure even more rapidly than they do in the superjacent parts. They almost always occur at the articular ends, but in the humerus, and to a less extent in the radius and ulna, exhibit a tendency to affect the shaft. They present themselves clinically as firm, elastic growths on one aspect of the bone, not expanding it, and not being covered by a bony shell; their base of attachment is large, and frequently osseous; the diseased bone may spontaneously fracture. The skin is often red, shiny, hot and tender; there may be effusion into the neighbouring joint, and the body temperature may be considerably elevated. The rapidity of growth differs in different cases, being generally more rapid in the round-celled tumours than in those which are composed of spindle cells, and whose stroma is often fibrous. If no operation be performed, the patient may die of the cachexia induced by the growth, or of secondary tumours in the viscera, especially in the lungs. If amputation of the limb be performed, the growth is exceedingly likely to recur,

FIG. 81.

Section of the Lower Half of a Femur with a Periosteal Sarcoma. The outline of the shaft can still be traced, but the cancellous tissue and the medulla have been infiltrated by the growth.

although, if the bone at the seat of section be healthy, recurrence is not frequent in the stump. The superficial lymphatic glands—*e.g.*, those in the inguinal region—often remain free from disease throughout; but the deeper ones—*e.g.*, those along the iliac vessels, or in the lumbar region—are somewhat more liable to be implicated by the secondary tumours. In some cases the viscera are largely involved.

An examination of the diseased limb will show a growth which infiltrates, but does not expand, the bone, and which is in no way limited by a capsule. The remains of the periosteum can often be traced, but in tumours of considerable size it becomes indistinguishable. The growth itself may either be a soft, pulpy, hæmorrhagic mass, or a gelatinous, opalescent, fleshy, and somewhat lobulated tumour; more rarely, it is fibrous on section. Mingled with the tumour is a variable amount of cartilage, bone, or calcareous matter. The bone is arranged in the form of delicate spiculæ or needles, standing out at right angles from the diseased shaft or articular end; this structure is best seen in macerated specimens, and is produced by the tendency to the formation of bone around the blood-vessels as they pass inwards from the periosteum.

Endosteal or central sarcomata grow almost invariably in the cancellous tissue of the articular extremities of the long bones, and hardly ever in the shafts. They are common also in the jawbones, and are met with, though less frequently, in those of the skull, carpus, and tarsus. Commencing in the cancellous tissue, they soon extend into and infiltrate the compact bone, and gradually expand it, and alter its shape. The thinned and expanded bone covers the growth like a shell, whilst from the periosteum new bone may be produced to supply the place of that which is destroyed. Spontaneous fracture may occur in consequence of the weakening of the bone. As the tumour increases, it bursts through its osseous capsule in places, and infiltrates the soft tissues. The articular cartilage offers a stubborn resistance to its progress, and is seldom involved in the

growth, although the latter may push it aside and reach the articular cavity. Endosteal sarcomata are of very variable consistence, and exhibit a great tendency to break down in their centres and form cystic cavities, which occasionally communicate directly with large blood-vessels, and are full of blood and tumour débris. The myeloid sarcomata are of a dark-red or maroon colour, and are more liable than others to develop cystic cavities.

Fig. 82.

Section of the Head of a Tibia, which has been expanded by the growth of an endosteal, myeloid sarcoma. The tumour has broken down and formed cysts.

Endosteal sarcomata present themselves clinically as growths expanding the bone, and not appearing on one side alone, as do the periosteal tumours. They are covered by a bony shell, which sometimes yields to pressure, and bulges or crackles beneath the finger; occasionally, pulsation is produced by the communication between the vessels and the cysts. The growth of endosteal sarcomata varies in rapidity, the myeloid tumours growing most slowly and the round-celled ones most rapidly. If no treatment be adopted, the patient may die either of the cachexia induced by the disease, or else from dissemination of the tumour. Glandular affection is rare. If completely removed by amputation or otherwise, myeloid sarcomata frequently do not recur either locally or elsewhere, and the tendency to recurrence and dissemination of all endosteal growths is much less than is that of the periosteal sarcomata.

Blood cysts in bone.—Most of the swellings of bone which on section are found to contain blood, are really myeloid sarcomata which have broken down, and the vast

majority of the so-called "aneurysms" of bone of the older writers undoubtedly belong to this class. Nevertheless, there are a few exceptional and rare cases in which simple blood cysts, quite innocent in their nature, develop in bone, and appear to consist of numerous thin-walled blood-vessels with a little surrounding connective tissue. Thus, I have seen a tumour which expanded the head of the tibia, and simulated a myeloid sarcoma, which on being opened was found to contain merely blood and clot. The cavity was subsequently gradually filled with granulation tissue, and now, five years later, there is no sign of any growth.*

TUMOURS OF THE JAWBONES.

Tumours of the jawbones possess so many distinctive features that they merit separate description. These distinctive characters depend partly on the proximity of the neighbouring mucous cavities, and partly also on the presence of the teeth and of the fœtal structures from which the latter originate.

Epulis.—The term epulis is often applied without distinction to any tumour which grows upon the gum, but is best limited to the "hard fibromata" which are common in this situation. The common fibrous epulis usually occurs in young people. It presents itself as a rounded, slightly pedunculated growth, seldom larger than a hazel-nut, and often smaller. It does not bleed except when it is ulcerated, and gives but little pain. Such tumours grow from the periosteum, and are occasionally attached to the fang of a tooth. Even when not so attached, their deeper part often extends into the neighbouring alveolus, and consequently their complete removal generally necessitates the extraction of a tooth and the excision of a portion of the alveolar margin.

The so-called "diffuse epulis" is merely a general thickening and hypertrophy of the tissue of the gum.

* See a paper by E. W. Roughton, Esq., read before the Med. Chir. Soc., December 10, 1889.

Sarcoma.—Myeloid sarcomata are often included amongst the epulides under the name of "myeloid epulis." They possess the characters common to endosteal sarcomata in other situations—growing within and expanding the jawbones, presenting as rounded, tense, and elastic swellings, often feeling suspiciously like cysts, and generally developing in young subjects. After complete removal they seldom recur, and never become disseminated.

In addition to the myeloid tumours, other varieties of central sarcoma occur within the jawbones, and are most frequently seen in the antrum. They are usually composed of round or oval cells, are very soft and succulent, of rapid growth, tending to extend into neighbouring cavities, to fungate through the skin, to affect the lymphatic glands, and to become disseminated.

As they increase in size, they expand the wall of the antrum on all sides. Thus, they thrust inwards the outer wall of the nostril, and obstruct respiration; they push up the floor of the orbit and cause protrusion of the eyeball; backwards, they extend to the pharynx; and downwards, they either cause the hard palate to bulge, or else make their way between the two layers of compact tissue of the alveolar process, and, after loosening one or more of the teeth, fungate through the gums. Any tumour growing within the antrum tends to distend the cavity in this way, but malignant tumours do so in a more typical manner than do the innocent growths. Glandular affection is not common in cases of sarcoma of the antrum, but does sometimes occur.

In addition to the myeloid tumours, fibro-sarcomata occasionally develop within the lower jawbone. These are sometimes hard and fibrous on section, and contain within their substance numerous osseous or calcareous granules. They are apparently not very malignant, and, when completely removed with the bone in which they grow, do not seem prone to recur.

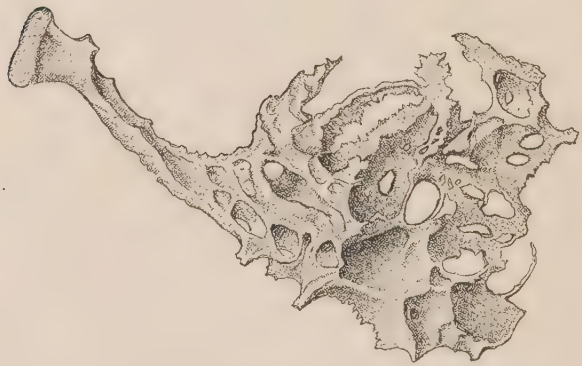
The periosteal sarcomata of the lower jaw do not present

any important points of difference from similar tumours on other bones. They are mostly of rapid growth, and are very malignant.

Carcinoma.—True carcinomatous tumours are met with in the antrum, developing in this cavity from the epithelium of its mucous lining. They grow in elderly people, and tend to run a rapid course, affecting the glands and becoming disseminated. Histologically, they are found to be epitheliomata with either squamous or columnar cells, or spheroidal-celled encephaloid cancers.

Multilocular cystic tumour.—The so-called multi-

FIG. 83.



A Lower Jawbone expanded and in part destroyed by a Multilocular Cystic Tumour.

locular cystic tumour appears to stand between the solid growths on the one hand and the true cysts on the other; on account of its structure, it is sometimes called "cystic epithelioma." These growths (which in this country have been especially studied by Mr. Eve*) are rare, and have until late years been very imperfectly understood, so that specimens of maxillæ dilated by cysts have been generally described as instances of true simple cystic disease of bone. It must not, however, be thought that in these tumours

* *Brit. Med. Journ.*, Jan. 6, 1883.

there is no new growth, for there may be so much as to give the appearance of a solid tumour, or so little as to make it appear that the jawbone is expanded merely by collections of dark, brownish fluid. Multilocular cystic tumours are most common in the lower jaw, and are of endosteal or central growth. They appear to originate from epithelial cells which have been originally involuted at the time of the formation of the teeth, and occasionally follow injury of the bone or caries of the teeth. The disease

FIG. 84.

Section of a Multilocular Cystic Tumour of the Lower Jaw. The matrix is fibrous, and contains masses of epithelial cells, which are breaking down in their more central parts. (Zeiss, A.)

may occur at any period of life, but is most common between the ages of twenty and forty. Growth is generally slow, and five or ten years may elapse before the patient applies for treatment. The tumours are not malignant, and show no tendency to affect the glands or to disseminate. They expand the jawbone, more especially on its inner side, and are frequently very hard and bony to the touch; they are occasionally elastic and crackling; the teeth become loosened, and fall out, and from their empty alveoli there may be a discharge of sticky fluid.

Section of the jawbone will show that the osseous structure is expanded, and that the cavities in it are filled, partly by a solid growth of soft fibrous or fleshy material, partly by fluid which is blood-stained, serous, or viscous. The septa which divide the cysts from one another are generally incomplete, and are either fibrous or osseous. The cysts vary in size from that of a split pea up to an inch or an inch and a half in diameter.

Microscopical examination shows that the new growth is composed of a fibrous stroma, in which are embedded columns or groups of epithelial cells. In the more central parts of these groups, the cells are to be seen undergoing mucoid or colloid degeneration, and it is by this destruction that the cavities or cysts are formed; the outermost cells in the columns are often cylindrical. It will thus be seen that these multilocular cysts are produced by an ingrowth of columns of epithelium, and it is by the subsequent disintegration of the cells which form the tumour that the cysts are developed. The latter are therefore secondary or degeneration cysts, and are strictly comparable with the degeneration cysts which are common in the endosteal sarcomata.

Periosteal cysts.—The periosteal cysts of the jaw are collections of fluid situated beneath the periosteum of the fangs. They seem to be of inflammatory origin, and are generally connected with carious teeth or with fangs left behind after imperfect extraction. They vary in size from that of a pea to a walnut, and when large, cause thinning of the superjacent bone. They are of common occurrence, and sometimes cause considerable pain.

Mucous cysts of the antrum.—Distension of the antrum by fluid is most probably in all cases the result of the formation of a cystic tumour in its lining membrane and not of distension by its own secretion. Such cysts are of a perfectly innocent nature, and may occur at all ages; they are not accompanied by, or dependent on, the development of any solid growth. In cystic disease of the antrum, this cavity is distended in the same manner as by a

malignant tumour, but there is no fungation, growth is not rapid, and as the bone is thinned, the presence of fluid becomes apparent.

Dentigerous cysts.—Dentigerous cysts are cavities containing serous fluid dependent upon impacted misplaced teeth—*i.e.*, they are associated with some impairment of the normal process of dentition, resulting in the retention of the tooth or teeth within the alveolar processs of the maxilla. With scarcely an exception, dentigerous cysts are formed in connection with the permanent and not with the milk teeth, and occur more frequently in cases of retention of the canine tooth than of any other. It by no means follows, however, that cysts result in all cases of retained permanent teeth.

Dentigerous cysts are formed by a distension of the tooth-sac with fluid secreted by the epithelium of the enamel organ. Consequently, the crown of the tooth itself projects into the cavity of the cyst, just as it formerly did into its own tooth-sac, and its fang is fixed into the sac-wall. As the cyst enlarges, it expands the jawbone, and forms a rounded, elastic swelling, the thin bony covering of which often crackles beneath the pressure of the finger. In cases of undue retention of the permanent teeth, the milk teeth are very late in being cast, and an examination of the mouth will therefore show either that a milk tooth is retained in that part of the jaw where the swelling is situated, or else, if the latter has been already shed, that a permanent tooth is missing. Although the retained tooth is at first adherent to the sac-wall, it becomes detached after a time, and may be found loose in the cavity. Dentigerous cysts appear to be most common in young adults, but have been met with in middle age.

Fig. 85.

Portion of a Dentigerous Cyst with a Tooth attached.

Diffused osseous growths.—The bony tumours which are found in the antrum are peculiar in this respect, that instead of growing from some one definite portion of the antral wall, they are diffused over the whole area of the latter. There is thus no definite tumour, but rather a general thickening of the whole of the bone which encloses the antrum, and a consequent gradual obliteration of the antral cavity by the new bone thus formed. But, although the increase of bone is mainly confined to the neighbourhood of the antrum, it extends after a time to the other portions of the superior maxilla, and may thus form a very considerable tumour. The growth of these osteomata is slow, and may extend over many years; their clinical course is entirely innocent; they are of rare occurrence, and are usually found in young people.

Leontiasis ossea is a disease characterized by a general overgrowth of the facial and cranial bones, with the formation of huge, rocky, masses of shapeless bone. The disease appears to commence in early life and its progress is slow, although, in the course of time, the cavities of the mouth, pharynx, or orbit may be encroached upon, and death may result from extension of growth to the cranial cavity. The cause of the disease is quite unknown.

Cartilaginous tumours of the maxilla are extremely rare; as in many other bones, the growth of the cartilage is usually associated with that of sarcomatous elements.

Odontomata, or tumours of the teeth, may be composed either of true bone or enamel. The latter never cause any symptoms, and are of little practical interest, but the exostoses of the fangs, as well as the tumours which caused the so-called "warty teeth," may be the cause of much trouble and pain, and may form very considerable growths in the jawbones. These warty teeth are subdivided into two varieties—the circumscribed and the diffuse dentinal odontomata. In the former, a portion of a single tooth is alone occupied by a small outgrowth. In the latter, the whole tooth is misshapen and faultily developed, its place

being taken by an irregular mass of dentine, enamel, and bone, whilst sometimes two teeth are fused. Tumours of this nature are very rare. The largest specimen hitherto described was as big as a turkey's egg, and another as large as a chestnut is on record. They grow within and expand the jawbone, and have hitherto been met with in the inferior maxilla only.

CHAPTER XXXVIII.

DISEASES OF THE SPINE.

SPINAL CARIES.—ANGULAR CURVATURE.—POTT'S DISEASE.

Caries of the spine, or Pott's disease, is a tubercular osteitis affecting the bodies of the vertebræ.

It occurs especially in scrofulous children, but is not limited to any age, and is frequently attributed to some injury. Any part of the spine may be affected, but the dorso-lumbar region is more often involved than any other.

The disease commences in most cases as "diffuse tubercular infiltration" of the cancellous tissue of the anterior part of the vertebral bodies immediately beneath the compact bone to which the intervertebral discs are attached. In other cases the disease appears to be primary in the intervertebral discs, and only secondarily extends to the bodies.

Sometimes the destructive process commences in a single vertebra, and extends from this as from a centre to the neighbouring vertebræ. In other specimens it is evident that the caries has been originally diffuse, and has implicated many parts of the spine at one and the same time.

The changes that occur in tubercular osteitis have already been described in detail. In the spine the process commences as a very insidious form of inflammation, accompanied by a growth of tubercle and lowly organised granulation tissue in the cancellous spaces. This is followed by rarefaction and absorption of the cancellous bone, and by caseation or

calcareous degeneration of the inflammatory products. By a continuance of this process, the body of the diseased vertebra is gradually destroyed, and the granulations extend into and destroy the intervertebral discs, and then involve the neighbouring vertebræ. It is to this gradual destruction of the bone that one of the most notable symptoms of spinal caries is due, namely, **angular curvature.** The weight of the head, shoulders, and trunk is transmitted to the lower extremities through the vertebral bodies, and it is evident that, if these soften and crumble away, the yielding and pulpy bones will be crushed together by the superimposed weight, so that as fast as the bone is absorbed by the granulation tissue just so fast are the diseased bodies and discs compressed. But the spinous, transverse, and articular processes are not destroyed; and if the anterior part of the spinal column, which is formed by the bodies, is shortened, whilst the posterior part, formed by the processes, is intact, it is evident that, as the bodies are compressed, the spinous processes must project backwards. In this way the angular curve is produced, the apex of the angle being formed by the spine of that vertebra which is nearest the centre of the destructive process.

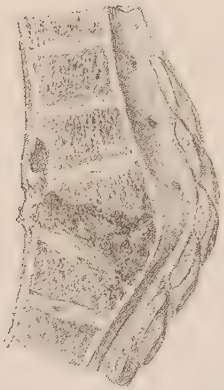
The further course of a case of Pott's disease depends much on the circumstances in which the patient is placed. When he is kept at rest and under good hygienic conditions the caries may stop, the inflammatory exudation may cease, the caseation of the granulation tissue may give way to organisation and formation of fibrous tissue, and the diseased vertebræ may be fixed to one another by fibrous adhesions or bony anchylosis, any angular curve which has been produced being thus rendered permanent. This reparative process is not limited to the vertebral bodies, but is aided by changes in the intervertebral articulations, which result in fixation of these joints by adhesions and in the formation in some cases of bony plates uniting the transverse processes.

In a large majority of cases no such favourable termination as that above described takes place, and the constant

rubbing of the diseased bones causes the progression and extension of the destructive process.

In such patients the granulation tissue breaks down into ill-formed caseous pus, which escapes through apertures at the front or sides of the carious vertebra, and in many cases strips the anterior common ligament from its attachment for a considerable extent. Very frequently the ligament effectually resists the pressure of the pus, and causes it to make its way laterally to the soft tissues at the sides of the diseased vertebra. From this situation it may either track backwards with the posterior branches of the intercostal or lumbar arteries and nerves, forming a dorsal or lumbar abscess, or may pass forwards into the tissues on the front and sides of the spine.

FIG. 86.



Spinal Caries. One intervertebral disc has been destroyed with the greater part of the vertebral bodies on either side of it. The anterior common ligament is partially separated. The spine presents a slight angular curve.

If such an abscess form in the cervical region, it may point behind the pharynx, may pass laterally between the muscles of the neck, may track downwards and enter the axilla with the axillary vessels and nerves, or in rare cases extend into the mediastina.

When the lower dorsal vertebræ are involved, the pus often tracks downwards, passes beneath the ligamentum arcuatum internum, and thus enters the sheath of the psoas muscle; or, in cases where the lumbar vertebræ are themselves diseased, the pus, escaping at the lateral margins of the bodies, is discharged directly into the fibres of the psoas which are attached to this part of the spine. In either case, the pus collects slowly in, and gradually tracks

along, the muscle until it passes with it from the abdomen to the inner side of the thigh.

When the pus misses the sheath of the psoas, it either enters that of the iliacus muscle, and points above Poupart's ligament, or else it passes down into the pelvis. From the pelvis it may escape by passing through the sacro-sciatic notch and pointing as a gluteal abscess, by tracking along the rectum into the ischio-rectal fossæ, or by bursting into one of the hollow viscera. Wherever the abcesses are situated, they are always at first chronic, and extend very slowly. In some rare cases, where treatment is rigidly carried out, the fluid portions of such pus may be absorbed, and the more solid parts may remain as a gritty or cheesy mass for many years. More commonly, after reaching a cutaneous surface, the abscess bursts, and, becoming contaminated by exposure to the air, a more acute inflammation of the abscess-sac ensues, pus is secreted more rapidly and in larger quantities, and suppurative or hectic fever, amyloid disease, or general tuberculosis bring about a fatal termination. In the larger number of discharging abscesses, small crumbs of dead bone may be found, and in many cases sequestra of considerable size are formed, which, being unable to escape from the seat of their formation, keep up a constant discharge of pus, and prevent sinuses from healing.

The spinal cord in most cases of angular curvature escapes compression for the reason that, the inter-articular joints being intact, the curvature does not cause a narrowing of the spinal canal. This, however, is not always the case, and in many specimens the calibre of the canal is distinctly diminished by a backward displacement of the softened bone at the seat of the greatest curvature. Such a narrowing, fortunately, does not necessarily imply interference with the functions of the spinal cord, and it is certain that the latter can indeed be considerably compressed without any symptoms arising, provided that the flattening is gradual, and not the result of sudden displacement. In some cases, nevertheless, **paraplegia** does occur, and appears to

be caused by an extension of inflammation to the meninges, with resulting pachymeningitis, or by the exudation of inflammatory products into the spinal canal. In the large majority of cases, moreover, the paraplegia is transient, and although in some it is rendered permanent by reason of structural changes in the cord itself, yet in most instances a good prognosis may be given if the patient is otherwise not too ill, and is placed under appropriate treat-

FIG. 87.

A Spine, showing an acute angular curve, with narrowing of the spinal canal and flattening of the cord.

ment. It should be added that, in cases of disease of the cervical spine, the danger of pressure on the spinal cord by displacement of the diseased vertebræ is much greater than in dorsal and lumbar caries, where the parts are steadied by the ribs, by the large trunk-muscles, and by the greater size of the opposed vertebral bodies.

LATERAL CURVATURE.

Lateral curvature of the spine is commonly the result of muscular weakness and overwork in growing girls and boys about the age of puberty, at a time when great demands are made on the strength. It is also caused by anything

which renders one leg shorter than the other—*e.g.*, old hip disease—the spine being obliged to curve in order to allow the foot to reach the ground. Some of the very worst cases result from an altogether different cause, namely, the falling in of the ribs which commonly follows empyema.

So soon as the muscles lose tone and cease to support the spinal column, the ligaments yield and allow the curvature to commence. The direction of the curve is to a great extent determined by the habits or occupation of the patient, but the most common primary deviation is seen in the upper dorsal region with the convexity to the right.

As soon, however, as the upper part of the spine curves to the right, the lumbar region develops a compensatory curve to the left, this being necessary in order to allow the patient to maintain the upright posture. At the same time that the lateral curvature occurs, the vertebræ also rotate, the rotation always being in one direction. The bodies are turned towards the convexity of the curve and the spines towards the concavity, so that the transverse processes towards the convexity are thus thrust backwards and made to project. The ribs necessarily follow the rotatory movements of the spine, and the shape of the thorax is altered in proportion to their displacement; the shoulder is thrust up, and the hip on the same side raised. In cases of long standing, the bodies of the vertebræ and the intervertebral discs become in time compressed on the side of the concavity.

CHAPTER XXXIX.

DISEASES OF JOINTS.

SIMPLE OR SEROUS SYNOVITIS.

A SIMPLE synovitis is an inflammation of the synovial membrane which is not dependent upon any constitutional disease or upon the introduction of septic material from a wound or neighbouring inflammation. This is one of the commonest troubles to which joints are liable, and affects particularly those articulations which are the most freely movable and the most exposed. The usual causes of simple synovitis are injury and exposure to cold or wet.

Acute synovitis.—The changes in the synovial membrane do not differ from those met with in inflammations of other soft tissues. At first there is intense hyperæmia, followed shortly by swelling, and exudation of fluid both into the membrane itself and into the articular cavity. The swelling is greatest in the situation of the normal folds; *e.g.*, in the ligamenta mucosa and alaria of the knee, which, by their increase in bulk, overlap the cartilages, and contrast strongly with the pearly-white colour of the latter.

Microscopically examined, there is seen to be much cell exudation into the perivascular spaces, with swelling and softening of the connective tissue by the excess of fluid in which it is soaked. The dilated capillaries occasionally give way, and thus cause minute extravasations of blood. The leucocytes escape into the substance of the membrane itself, and also penetrate between the endothelial cells and reach

the articular cavity. The endothelial cells appear to multiply with unusual rapidity, and are liable to be cast off into the joint in considerable numbers.

Changes in the synovial fluid.—In the early stages of an acute synovitis, the synovial fluid is simply increased in quantity, but, as the inflammation progresses, the normal secretion is mingled with serum in varying proportions, and afterwards with the fibrin-forming elements of the blood, and with red and white corpuscles. At first the fluid is clear, but later on it becomes cloudy, opalescent, or blood-stained. In cases where the original injury has been severe, there are often considerable extravasations of blood into the articular cavity. In most cases the blood does not clot for some time, probably on account of its admixture with the serum and synovia, as well as because of the smooth endothelial lining with which it is in contact.

If a simple acute synovitis undergoes resolution, the exudation of cells ceases, the vascularity subsides, the exuded fluid is absorbed, and the membrane and its secretion again present a natural appearance. When there is much extravasated blood, resolution is generally greatly rolonged.

Subacute and chronic synovitis.—If a joint which is the seat of an acute synovitis be not kept at rest for a sufficient length of time, the inflammatory process is liable to pass into a subacute or chronic stage, and the absorption of fluid from the articular cavity ceases. In other cases the inflammation, from the beginning, is of but slight intensity.

In this form of synovitis there is little hyperæmia, but often a good deal of swelling, and where the inflammation is of long standing the membrane itself is liable to be much thickened by the formation of fibrous tissue.

The fluid in the joint is generally greatly in excess of what is natural, and consists chiefly of serum. It is usually quite clear, but may contain small shreds of fibrin or the melon-seed bodies which are more commonly seen in bursæ.

The long-continued distension of the joint is liable to result in the stretching and weakening of its capsule and ligaments. The terms "hydrarthrosis" and "hydrops articuli" have been applied to the more chronic forms of synovitis with effusion.

ACUTE SUPPURATIVE ARTHRITIS.

Acute suppurative arthritis is a general infective inflammation of all the structures which enter into the formation of a joint. It may be produced in one of the following ways:—

1. By a wound which opens the articular cavity.

2. By extension of inflammation from the articular bone, especially by tracking of pus in cases of acute periostitis, or by the rupture of an abscess in the bone.

3. By extension of suppuration from the soft tissues. This is rare in cases of simple suppuration, but may result from sloughing of the tissues over a joint after an injury, from phlegmonous erysipelas, or from extension of suppuration from a bursa which communicates with the articulation—*e.g.*, the bursa beneath the psoas.

4. As a complication of various forms of blood-poisoning, especially pyæmia, puerperal fever, and more rarely typhoid fever, scarlatina, gonorrhœa, &c.

The synovial membrane is the first of the articular structures which shows signs of inflammation. It becomes at first bright red and swollen, and in a very short time it loses its polished appearance, and is covered with shreds of adherent fibrin. If the latter are peeled off, the synovial surface is found to be rough and velvety, like granulation tissue. The synovial fluid is increased in quantity, soon becomes blood-stained and opalescent, and very shortly is mingled with pus which has been formed by the inflamed synovial surface.

Within a day or two the inflammatory process extends from the synovial membrane to the cartilages, and the latter lose their pearly-white colour, are permeated by new blood-

vessels, and ulcerate in patches, or necrose and are cast off in shreds.

The ligaments share in the general suppuration; their dense structure is split up by infiltration of serum and pus, and they become ragged and shreddy, yield, and allow displacement of the articular surfaces. The bones are not spared. The cartilages being destroyed, osteitis ensues, and the usually smooth layer of articular bone becomes rough and carious. The peri-articular tissues share in the general suppuration, and abscesses form around the joint. In many cases the suppuration in the tissues is the result of the yielding of the softened and distended capsule, which thus allows the sudden escape of its contents. This is a fact of much clinical importance, for, when the pus escapes from the joint, the swelling and pain in the latter often partially subside, and, unless the surgeon be on the watch for it, he may overlook the pus outside the articulation. In cases of acute suppuration of the knee, collections of pus may extend amongst the muscles of the thigh almost as high as the hip-joint.

The microscopical appearances in suppurative arthritis do not differ from those ordinarily met with in acute inflammations of bone and soft tissues in other parts of the body, and require no special description.

Acute suppurative arthritis is accompanied by much redness and swelling of the inflamed joint, by great pain, and severe constitutional disturbance. If not properly treated, it may terminate fatally, and, in many cases, even when by free incisions the pus has been satisfactorily evacuated, amputation has subsequently to be resorted to on account of the exhaustion which follows the discharge of large quantities of pus, and the accompanying suppurative fever.

If recovery ensues, the joint is usually left in a state of bony anchylosis, and, this being the case, it is evidently of the utmost importance to see that the limb is kept in as good a position as possible. In such cases, after the pus

has been evacuated and the acuteness of the inflammation has subsided, the suppuration gradually ceases. The cells which have been exuded into the substance of the synovial membrane, cartilage, bone, &c., are gradually developed into fibrous tissue, in which, where it is in contact with osseous tissue, bone salts are subsequently deposited. The hyperæmia subsides; the fibrous tissue, which is not ossified, shrinks; the cavity of the joint is obliterated; and the articulation, as such, ceases to exist.

In favourable cases, and especially in children, when the pus has been early evacuated, a more or less movable articulation may remain, though, at best, such a joint is permanently weak.

ACUTE ARTHRITIS OF INFANTS—ACUTE EPIPHYSITIS.

These names are applied to cases of acute suppurative arthritis occurring in infants and children, and resulting from inflammation of the articular bone. Attention has been specially directed to this subject by Mr. T. Smith.

The patients who are the subjects of this form of disease are seldom more than a year or eighteen months old. The joints most commonly affected are the hip, knee, shoulder, ankle, and elbow.

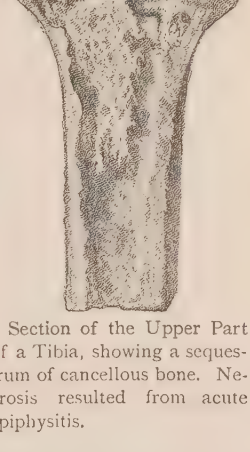
The clinical course is rapid, and does not differ materially from that of acute suppurative arthritis due to injury. In some cases there is a history of a blow or other injury; in others, none can be obtained.

The disease appears to commence as an acute inflammation of the most recently formed bone around the centres of ossification, and commonly results in necrosis and the formation of a minute sequestrum. Around this, pus is formed, and subsequently makes its way into the neighbouring articular cavity, the aperture by which the pus escapes into the joint from the epiphysis being often so minute that the real cause of the arthritis is liable to be overlooked unless a section is made of the articular bone. This escape of the

pus is followed by acute arthritis and the formation of periarticular abscesses.

Many cases terminate fatally, but, if an early exit be given to the pus, the patient frequently recovers. In some instances the joint, in spite of the acuteness of the inflammation, suffers comparatively little permanent injury. In others, which are unfortunately the more common, the growth of the bone is seriously affected, and the articular extremity is permanently deformed. In such cases the joint is liable to be extremely loose and flail-like, and, in the case of the lower extremity, is unable to support adequately the weight of the body.

FIG. 88.



Section of the Upper Part of a Tibia, showing a sequestrum of cancellous bone. Necrosis resulted from acute epiphysitis.

Acute epiphysitis is not, however, limited to infants, for cases of a precisely similar nature are not infrequently met with in older children and in young adults, and run a very similar course. The extent of the necrosis in these cases is nevertheless liable to be much greater than in infants, and sequestra of considerable size may be formed. The tendency of the pus to extend into the neighbouring joint and to cause acute arthritis is unfortunately as great in these cases as in the younger patients, and the prospects of recovery with a movable articulation are by no means so good.

PUERPERAL ARTHRITIS, AND ARTHRITIS FOLLOWING THE EXANTHEMATA.

A common complication of the puerperal condition is inflammation of one of the large articulations, and especially of the knee. In such cases there may be merely a simple

transient synovitis, but in many others there is a more chronic inflammation, and a tendency to fibrous anchylosis. In others, again, the inflammation progresses to suppuration, and is often associated with necrosis of some of the articular bone, *e.g.*, of the lower end of the femur. The course run by these latter cases has already been described under the head of "Acute Suppurative Arthritis."

Of all the exanthemata, scarlatina is more frequently followed by inflammation of the joints than is any other. In cases otherwise uncomplicated there is frequently a subacute synovitis of several articulations, resembling closely in its clinical characters the synovitis of acute rheumatism, and running a similar course. In those cases of scarlatina, however, which are complicated by parotid buboes, sloughing of the tonsils, &c., there may be a genuine pyæmic arthritis, which runs the course typical of such a disease. Mumps, dysentery, and measles may be followed by similar inflammation of the joints. Typhoid fever and, more rarely, smallpox may also be followed by disease of the articulations.

In many cases, and especially after typhoid, one joint alone is attacked, and of all joints the hip is that most frequently implicated. The character of the inflammation differs in different cases. In some there is a simple synovitis; in others, a more general arthritis, terminating in anchylosis more or less complete; in others, again, there is acute suppurative arthritis. The hip-joint is liable to be affected in a somewhat peculiar manner, for in many of these cases there is a very rapid effusion into the synovial cavity, accompanied by softening and stretching of the ligaments, and followed by **spontaneous dislocation** of the femur on to the dorsum ilii. All this may occur without the formation of any pus.

URETHRAL ARTHRITIS (GONORRHŒAL RHEUMATISM).

The term urethral arthritis is applied to cases of inflammation of joints following urethritis, and is preferable to

the older one of "gonorrhœal rheumatism," for it may complicate cases which are not of venereal origin, and, although more common in males, is seen in some cases of simple leucorrhœa in women. Certain individuals seem especially predisposed to this form of arthritis, which appears to attack by preference those who are of a gouty or rheumatic diathesis. The real nature of the affection is still unsettled, but by most writers it is considered to be the result of absorption of infective material from the urethra. Others look upon the disease as of neurotic origin, and think that it is excited reflexly. In urethral arthritis one large joint—and that most commonly the knee—is usually affected, but no joint is altogether exempt, and the fasciæ in the sole of the foot may be implicated, and may, by their yielding, lead to the production of flat foot.

The disease appears to be essentially an inflammation of the synovial membrane and ligamentous structures, and is very liable to be extremely chronic, to recur occasionally, and to terminate in fibrous anchylosis. More rarely, suppurative arthritis supervenes, and runs a course such as has been already described.

PYÆMIC ARTHRITIS.

In most cases of pyæmia one or more joints become inflamed. In many cases the inflammation is limited to the synovial membrane, and the joint is quickly filled with a mixture of synovia, serum, and thin, yellow oily pus. In several such instances in which I have made post-mortem examinations, the joint, after having been washed out, presented no signs of inflammation, save a slight swelling of the synovial membrane, and no signs of ulceration of cartilages or bones could be seen. In cases such as these the effusion may be absorbed, and the joint may then either return to its natural condition or may be partially anchylosed by the formation of fibrous adhesions. I have on several occasions seen joints become quite stiff after pyæmia without the occurrence of any suppuration. This is not

however, always the case, for sometimes the pus is produced in greater quantities, the inflammation spreads to the cartilages, bones, and ligaments, and a general suppurative arthritis ensues.

GOUT.

Gout is a constitutional disease, characterised by inflammation of the joints, with deposit of urate of soda in the articular structures. The general pathology of gout is beyond the scope of the present work, and the changes in the articulations cannot be described at length.

The patients most subject to gout are those of the uric-acid diathesis, and are commonly past middle age. The joint most often affected is the first metatarso-phalangeal, probably for the reason that it is damaged more frequently than any other by pressure of ill-fitting boots, &c. No joint is exempt from attack. Gout is commonly said to be a disease of the rich, and not of the poor, but this is certainly incorrect, and in a very considerable proportion of post-mortem examinations of hospital patients over fifty years of age I have found urate of soda in one or more articulations.

The affected joints are liable to attacks of acute inflammation, but in many cases a joint appears to become the seat of gouty deposit without any history of an acute arthritis.

The morbid changes are chiefly as follows:—In any acute attack the synovial membrane presents the appearances common to all cases of simple synovitis, and the synovial fluid is similarly changed in amount and character.

The cartilages are inflamed, their matrix fibrillates, and their cells multiply. In the fibrillated cartilage, and on its surface, a white deposit of urate of soda ensues, and in subsequent attacks, the cartilages become more and more fibrillated and worn away, and the urate of soda increases in quantity.

When the bones are exposed, they, in their turn, become

the seat of gouty deposit, and not only they, but the ligaments, the synovial membrane, peri-articular connective tissues, and bursæ are similarly affected.

It is commonly stated that the urate of soda is simply deposited on cartilages which are otherwise normal, but my own experience is that this is not the case, and that wherever there is urate of soda there the cartilage is generally fibrillated and eroded. It appears probable that some at least of the urate of soda is formed by disintegration of the cartilage itself, and that it is not simply deposited independently of antecedent changes in the cartilaginous matrix. In very many cases, joints which are the seat of gouty arthritis present many of the changes which characterise osteo-arthritis. When the deposit of urate of soda is considerable, it may extend into the neighbouring tendon-sheaths, and, still increasing, may cause ulceration of the superjacent skin. In such cases, the protruding mass is called a "chalk stone" or "tophus"; these are more common in the fingers and ears than elsewhere.

RHEUMATISM.

The subject of acute rheumatism is one of medical rather than of surgical import, and the changes in the joints alone will be considered here.

In many cases there is nothing more to be seen on examination than in simple serous synovitis, but in acute rheumatism there is a definite tendency for the inflammation to extend to the sub-synovial and peri-articular tissues and for the synovial secretion to be more fibrinous, and consequently more shreddy, than in simple synovitis. In severe cases the cartilages acquire a bluish or opalescent tint, and may be distinctly swollen; in such instances, microscopical examination shows cell proliferation and exudation, and in a few cases the surface becomes fibrillated or eroded. The ligaments appear to be comparatively frequently implicated, but the inflammation rarely extends to the bones.

The ordinary duration of the synovitis in a simple case in any individual joint, varies from about three days to a fortnight, but in more severe cases, where the deeper structures are implicated, the inflammation often drifts into a subacute or chronic stage. As a rule, most patients recover without any permanent joint lesion, but it will easily be understood that, where the ligaments and peri-articular structures have been involved in a plastic inflammation, permanent stiffness or complete fibrous anchylosis may result. This does not often occur in more than one joint. In rare instances even suppuration may ensue.

The term **chronic rheumatism** has been very vaguely applied. It is better not to use it as synonymous with osteo-arthritis, but to limit it to cases in which, after one or more attacks of acute rheumatism, a chronic synovitis, with thickening of the ligaments and peri-articular structures by fibrous adhesions, supervenes. It may affect one or more joints, and frequently terminates in fibrous anchylosis.

CHAPTER XL.

DISEASES OF JOINTS—(*continued*).

OSTEO-ARTHRITIS.

Osteo-arthritis is a form of chronic joint disease characterised by peculiar degenerative changes in the articular structures, which do not tend to terminate in either anchylosis or suppuration. Other common names for the same disease are "rheumatic gout," "rheumatoid arthritis," "chronic rheumatic arthritis," "arthritis deformans," and there are many more.

The morbid anatomy of osteo-arthritis is very striking, and in well-marked instances the pathological changes cannot be mistaken for those of any other disease.

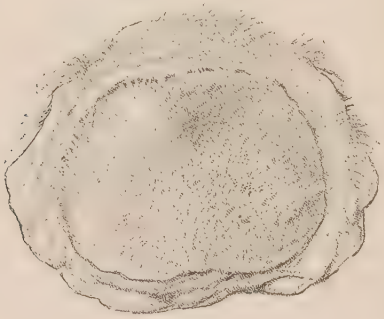
The disease may begin either in the synovial membrane or in the cartilage, and, judging by my own experience, which has been derived from an examination of many joints in the post-mortem room, I should say that changes in the cartilage almost always precede those in the synovial membrane.

Changes in the cartilage.—The first thing that is noticed is a slight roughening of the normally smooth cartilage, which, when subjected to a gentle stream of water, is seen to be broken up into delicate fibrils, arranged with their long axes at right angles to the articular surface, and resembling the pile of coarse velvet. This change is always most noticeable at those parts which are subject to the greatest pressure or friction, whilst at the margins the cartilage hypertrophies, and forms irregular nodular out-

growths, or "**ecchondroses.**" These outgrowths, at first cartilaginous, soon become bony in their deeper parts; the ossification extends through their whole thickness, and the "nodular osteophyte" thus produced is fixed more or less firmly to the subjacent bone, the articular borders of which are thus rendered prominent or "lipped." Sometimes these nodular masses are broken off, and form loose bodies in the joint, or in the substance of the synovial membrane itself.

As the disease progresses, the fibrillated and degenerate

FIG. 89.



A Patella from a case of Osteo-arthritis, showing fibrillation of the cartilage.

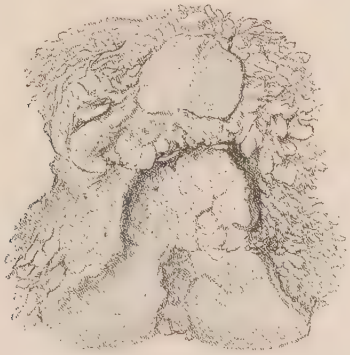
cartilage, no longer able to resist the attrition to which it is subjected by the movements of the articulation, is slowly worn away in patches, and the subjacent bone is exposed.

A microscopical examination of the cartilage in this stage throws much light on the progress of the disease. The normal hyaline matrix is broken up into fibres, in the midst of which the cartilage cells are found to be arranged in vertical columns whose long axes are at right angles to the articular surface. The cells proliferate and distend their capsules until they burst, with the result that the cells nearest the surface are cast loose into the synovial cavity. In this way the surface of the cartilage is broken

up, and the fibrillated matrix between the rows of cells remains to form the longitudinal striæ and tufts which give the velvety appearance already described.

At the margins, where the ecchondroses are formed, the same proliferation of cells and fibrillation of the matrix ensue, and it has been suggessed by Cornil and Ranvier that the heaping up of new cartilage in this situation is to be explained by the fact that the edge of the cartilage is covered by a prolongation of the synovial membrane; the

FIG. 90.



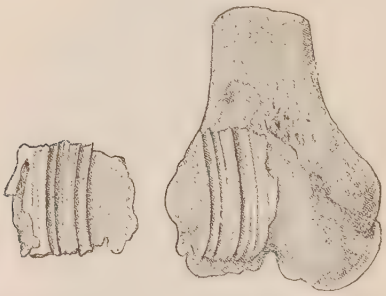
Femur, Patella, and Synovial Membrane from a case of Osteo-arthritis. The articular surface of the femur is deformed by the formation of large ecchondroses, and the synovial membrane is covered by hypertrophied synovial fringes.

cells, consequently, instead of escaping into the synovia cavity, are retained in the sub-synovial tissue, and by their constant multiplication produce the cartilaginous outgrowths. In some cases these ecchondroses protrude the synovial membrane in front of them, and at length project through it, so as to become intra-articular. In other instances they grow laterally, and do not encroach upon the joint.

The synovial membrane in the early stages of the disease shows little change, but after a time becomes

increased in vascularity, as well as thicker and tougher. In the later stages its fringes increase in size, their villous tufts hypertrophy and subdivide, and gradually the whole membrane assumes a shaggy or villous appearance. These enlarged villi are chiefly fibrous in structure, but often contain a little fat. In other cases cartilage is developed in them, and more rarely they become calcified or ossified. As they increase in size they get more pedunculated, and occasionally are completely detached, thus forming one of the varieties of "loose bodies in joints."

FIG. 91.



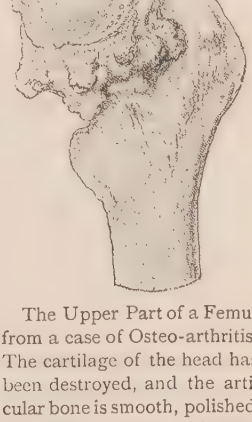
Femur and Patella from a case of Osteo-arthritis.
Both bones are much deformed, and marked by deep grooves.

The synovial fluid in the early stages of the disease is commonly increased, frequently to a very great extent; it is usually more cloudy and tenacious than is natural. As the arthritis advances, the secretion is liable to diminish, and to become still more thick and viscid.

The changes in the bones are very characteristic. The cartilage being worn away, the articular bone is exposed, and, in consequence of the friction to which it is subjected, becomes smooth and polished. The subjacent cancellous tissue also undergoes rarefaction and atrophy, with the result that the degenerate osseous tissue is quickly worn away in the movements of the diseased joint. The surface bone, by reason of the attrition, becomes smooth and

polished, ivory like or eburnated, and is usually not worn evenly, but in grooves. These are to be attributed to the rubbing of the opposed bone-lamellæ, which are arranged at right angles to the articular surface, and tend to scrape the opposing bone as might the teeth of a comb. As a rule the cancellous bone is not exposed by the atrophic proceess, for as the surface layer of smooth bone is worn away it is as constantly reproduced. This, however, is not always the case, and sometimes the enlarged Haversian canals may be seen opening on to the articular surface, and giving the bone a "worm-eaten" appearance. In some cases there is also a formation of very dense and porcelain-like bone, which is met with in no other form of joint disease. It is developed where there has been much friction, and, on account of its white appearance, is called "porcellanous."

FIG. 92.



The Upper Part of a Femur from a case of Osteo-arthritis. The cartilage of the head has been destroyed, and the articular bone is smooth, polished, and worn down. Several nodular osteophytes have developed on the neck.

But whilst these changes are in progress where the bone is exposed to friction, other changes ensue at the articular margins. Here, as has already been described, the nodular osteophytes are developed from the cartilaginous outgrowths, and by a continuation of the wearing away of articular bone and the production of these bony "lips," the shape of the articulating surfaces become profoundly changed. Thus, in the hip, the head of the femur is worn away and flattened, whilst from the margins of the articular cartilage nodular growths arise. In the course of time the whole head may be absorbed, and the articular surface may be formed by the polished stump of the neck. In the acetabulum, the

floor is polished and smooth, and the margins worn away and overgrown by new bone, so that the shape and even the position of the cavity are quite altered.

Osteophytes are also, but more rarely, developed at the points of insertion of muscles, or in the capsule close to its attachment to the bone.

The ligaments share in the general destruction, and like the other tissues, seem rather to degenerate and wear away than to be destroyed by any active process. If examined when fresh, their texture is seen to be more loose than natural, and their surface presents a ragged or frayed appearance. On account of their softened condition, they tend to allow the articulating surfaces to be displaced and loosened.

In many cases the **tissues outside** the affected joints suffer. Thus, the neighbouring muscles waste, the subcutaneous tissues become œdematous, and the skin is often shiny and smooth. Tendons also in the near neighbourhood fibrillate and wear away, just as do the intra-articular ligaments. This change is best seen in the shoulder-joint, where the part of the long tendon of the biceps which lies within the capsule is usually found either thinned and flattened or else entirely absorbed.

The patients who are the subjects of osteo-arthritis are usually over middle age, but the young are not always spared. Sometimes only one large joint, and that by preference the hip or the shoulder, is affected: in other cases several articulations are attacked, and, when the knee is implicated, the disease is commonly symmetrical. In the case of the hands, all the finger-joints are liable to be diseased. However long osteo-arthritis lasts, it never causes true anchylosis, except when the vertebræ are involved. It frequently results in great and serious impairment of the mobility of the joints, but this is explained by the alterations in the shape of the articular surfaces, by the destruction of the cartilage, and by the thickening of the synovial membrane. In other cases undue mobility and

laxity result from destruction of ligaments and wearing down of the articular bone.

Osteo-arthritis is usually very chronic and slow in its progress, but is occasionally of tolerably acute onset, and progresses rapidly. When occurring in early life, it is more liable to attack many joints than when it commences in old age.

Osteo-arthritis appears to follow attacks of acute rheumatism in a very small percentage of all cases, and certainly is most common in patients of a rheumatic or gouty descent. In many instances it is apparently to be attributed to frequent exposure to cold and wet, as well as to insufficient nourishment. In the case of the hip, it is certainly sometimes induced by an injury, especially by a fall on the trochanter. This is a fact of much clinical importance, and affords a ready explanation of those cases where shortening and eversion of the thigh follow within a few months of a fall on the hip. When the result of injury, osteo-arthritis appears often to progress with unusual rapidity, and the deformity it causes may easily be mistaken for that which results from an impacted intra-capsular fracture.

The true pathology of osteo-arthritis is still obscure. In its nature it seems to be more degenerative than inflammatory, but whether it is the result of an inherited or acquired diathesis, whether an expression of gout or rheumatism, or whether, as is supposed by some, of neurotic origin, is at present a matter of doubt.

CHARCOT'S DISEASE—TABETIC ARTHROPATHY.

Charcot's disease, as it is met with in this country, is a form of arthritis, allied to osteo-arthritis, which is developed in connection with tabes dorsalis. It is met with in but a small percentage of cases of tabes, and often commences when the tabetic symptoms are but little marked, or even unnoticed by the patient; very frequently it occurs before there is any evidence of ataxic gait. As described by

Charcot, the disease is usually of very sudden onset, the affected articulation becoming distended with effusion within twenty-four or thirty-six hours, without any apparent cause. This swelling of the joint is often accompanied by swelling of the neighbouring soft tissues, which do not, however, pit on pressure to any extent. In some instances the effused fluid is absorbed, and the joint returns to its natural condition; but in others, and these unfortunately are the more common, the effusion is but the commencement of a series of changes, which rapidly terminate in the destruction of the articulation. Within a few weeks or months from the first attack the patient notices that the joint gets weaker and gives way under him, and, in a very short time, undue mobility, with the production of a "flail joint," movable in all directions, or else actual dislocation ensues. One of the most noticeable of the clinical features is the entire absence of pain throughout the progress of the case.

If such a joint be examined, it will be found that the morbid appearances are very similar to those met with in osteo-arthritis. But just as Charcot's disease differs in its clinical course from osteo-arthritis in the rapidity of the destruction and the exceeding mobility, or even dislocation, of the articulation, so in its anatomical aspects it differs in the extent of the destructive changes rather than in their character.

The synovial membrane and its secretion show the same appearances which have been described as occurring in osteo-arthritis, but the synovial fluid is almost always greatly increased in quantity.

The cartilages fibrillate and wear away, and from their margins spring ecchondroses. The ligaments also fibrillate, stretch, and waste.

The most characteristic changes are found in the bones. These, in typical cases, are worn down to an extent never seen in osteo-arthritis, and are moreover often simply worn down without any new bone being produced, as is common in the latter disease. In typical cases the whole of the

head of the femur or humerus, the condyles of the femur or the head of the tibia, are ground down and destroyed as if they had been rubbed away by a grindstone or a file. It is this extensive destruction of bone, even more than the wearing away of the ligaments, which must be held accountable for the dislocations and undue mobility above mentioned.

FIG. 93.

Knee-joint from a case of Charcot's disease, showing the excessive wearing away of the bones and the displacement of the articular surfaces.

FIG. 94.

Another view of the bones depicted in the last figure, showing the complete wearing away of one of the femoral condyles.

It is evident that bones which wear away in this manner must be in a state of advanced degeneration, and to such an extent are they sometimes degenerated that spontaneous fracture ensues, a complication which never occurs in simple osteo-arthritis.

From the above description it would appear that Charcot's disease is justly separable from osteo-arthritis, and that typical specimens of the one can be easily recognised and differentiated from those of the other. It would, however, be a grave error to suppose that this is always so, for both clinically and pathologically numerous connecting links may

be found, and I have seen tabetic patients in whom the joint disease has lasted for several years, and whose articulations after death were not to be distinguished from those of osteo-arthritis.

There has been much discussion as to the true nature of Charcot's disease, but although the question is yet far from settled, the general opinion is that it is directly dependent upon degeneration of either the spinal cord or of the peripheral nerves, for the latter have been shown to be affected in cases of tabes. If this be true, the joint affection must be classed as a neurosis, and thus becomes strictly comparable with the perforating ulcers of the foot, which are also common in patients with sclerosis of the posterior columns of the cord. Some surgeons, however, look upon Charcot's disease as identical with osteo-arthritis, and consider that any differences are explained by the fact that the disease occurs in a patient affected by tabes, whilst others consider that the disease of the spinal cord and of the joints may each be the result of some common cause.

CHAPTER XLI.

DISEASES OF JOINTS—*(continued)*.

LOOSE BODIES IN JOINTS.

The loose bodies found in joints vary both in structure and origin. They may occur in articulations otherwise healthy, or may complicate osteo-arthritis and chronic synovitis.

Loose bodies may be formed in the following ways :—

First, as the result of an injury a portion of synovial membrane sometimes becomes thickened and indurated. It is probable that this condition originates in a rent or tear of the membrane, which is subsequently pinched and dragged upon in the movements of the joint, and is kept in a state of chronic inflammation. Bodies of this nature consist of fibrous tissue, fat, and inflammatory products. They are attached by pedicles, which tend to become longer and thinner the longer the growths exist.

Fig. 95.

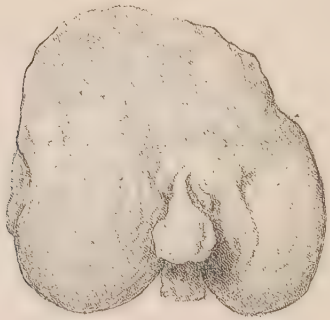
A Thickened Portion of Synovial Membrane, which formed a loose body in the knee-joint.

Secondly, in osteo-arthritis, as already described, the synovial membrane is liable to become covered more or less thickly with pedunculated growths springing from the normal fringes, and consisting of fibrous tissue or cartilage.

These bodies, although at first attached, may be separated and cast loose into the synovial cavity.

Thirdly, in osteo-arthritis the ecchondroses which spring

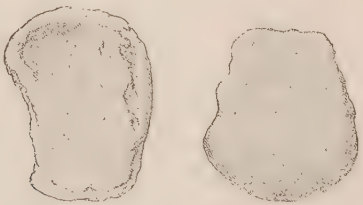
FIG. 96.



The Condyles of a Femur from a case of Osteo-arthritis, showing a nodular osteophyte merely attached by a slender pedicle of synovial membrane.

from the margins of the articular cartilage, and consist of cartilage and bone, may become detached, and either remain in the substance of the synovial membrane itself or else become loose in its cavity.

FIG. 97.



Two Portions of Articular Cartilage, apparently from the Femoral Condyles. They were removed by operation, one from the right knee, the other from the left.

Fourthly, in some cases portions of the articular cartilage are found as loose bodies. It is supposed that they may become separated either directly by the application of

violence, or may exfoliate as the result of some impairment of their vitality by an injury. Loose bodies may also be formed by the partial detachment of one of the semilunar cartilages.

Fifthly, in cases where there has been effusion of blood or of inflammatory exudation, masses of fibrin and blood-clot may remain in the synovial cavity.

Sixthly, blood may be effused into the substance of a synovial fringe, and may subsequently be "organised" into a hard fibrous lump.

Seventhly, in one case a loose body was found to have been formed around the point of a broken needle.

Eighthly, loose bodies are occasionally of tubercular origin, and may be multiple. I have seen five in one knee-joint. They may consist either of fibrin alone or of fibrin covering a tubercular fringe which has become detached.

Some of these modes of formation of loose bodies are of great rarity, and it is quite certain that the large majority of specimens are derived in the manner described under the first three heads. In the absence of injury, it appears probable that osteo-arthritis must be held responsible for the larger number.

If a loose body is left in a joint, free to move about without restraint, it commonly sets up a chronic synovitis, and, through causing sudden wrenches of the articulation whilst in use, often brings on an attack of acute synovitis. The usefulness of the limb is sometimes seriously impaired.

Of all joints, the knee is the one in which loose bodies are most frequently found, but the hip, the shoulder, and the elbow are occasionally affected.

Internal derangement.—The subject of internal derangement is one which may be briefly dealt with in connection with that of loose bodies in joints, for there is some similarity in the symptoms caused by these two affections. The term is applied to cases in which a joint, especially the knee, occasionally becomes suddenly locked or

fixed so as to temporarily prevent all movement. It is now tolerably clear that this condition is generally the result of an injury to the ligaments which fix the semilunar cartilages, resulting in their displacement when any strain is suddenly thrown upon the limb. Beyond the inconvenience of such attacks, and the synovitis which follows them, no ill results ensue.

JOINT DISEASE IN HÆMOPHILIA.

In the disease known as hæmophilia, or the hæmorrhagic diathesis—*i.e.*, in patients who are commonly known as "bleeders," the joints are liable to become temporarily swollen after slight injuries, and, after many such swellings, sometimes become stiff. Specimens of joints affected in this way are very rarely met with; but in two cases which occurred in St. Bartholomew's Hospital, in which death resulted from prolonged hæmorrhage following slight skin wounds, an opportunity was afforded of examining many of the articulations. These specimens were described by Dr. Wickham Legg at meetings of the Pathological Society in 1881 and 1885,* and allusions were made by him to two other cases which have been recorded.

FIG. 98.

Knee-joint from a "Bleeder." The synovial membrane is stained with blood, and a roughened spot in the outer condyle of the femur indicates the place where the patella was fixed by fibrous adhesions.

It would appear that the swellings of the joints are the result of hæmorrhages, and that, either from the irritation

* See vol. xxxiii. p. 412, and vol. xxxvi. p. 488.

caused by the frequent presence of clot in the joint or from the constitutional condition of the patient, the cartilages and synovial membrane undergo further changes. The former fibrillate and break up on their free surfaces, and become lipped at their margins as in osteo-arthritis, whilst the synovial membrane remains more or less stained by blood, and thickened by fibrous tissue. In one of the joints in the museum of St. Bartholomew's Hospital—a knee—there had further been a tough fibrous adhesion formed between the under-surface of the patella and the condyle of the femur (see Fig. 98). The ligaments were not noticeably affected. The bones were healthy, and there was no appearance of ulceration of the cartilages, or of such pulpy swelling of the synovial membrane, as is usual in strumous disease. It is probable that similar conditions are common in the joints of bleeders, and would evidently satisfactorily account for the symptoms met with in such patients.

SYNOVIAL CYSTS IN CONNECTION WITH JOINTS.

The occurrence of large cysts containing synovial fluid in connection with joints was first noticed by Mr. Morrant Baker, and recorded by him in vols. xiii. and xxi. of the *St. Bartholomew's Hospital Reports*. Since the publication of the first paper twelve years have elapsed, and during that time many other cases have been observed, in several of which I have had opportunities of examining the joints after removal. Some of these have been described by Mr. D'Arcy Power in papers in the Pathological Society's *Transactions*.*

From a consideration of the various recorded cases and dissected specimens, it seems safe to draw the following conclusions:—

First, a cyst may exist in connection with a joint which is itself perfectly healthy, but if the cyst inflame, as the

* Vol. xxxvi. p. 337, and vol. xxxvii.

result of injury or of surgical treatment, disease of the articulation may secondarily result.

Secondly, in cases of osteo-arthritis and of Charcot's disease, cysts may form, and it is probable that they are more common in connection with these affections than under any other circumstances.

Thirdly, synovial cysts may complicate strumous or tubercular disease of joints.

FIG. 99.

Posterior View of a Normal Ankle-joint injected with Gelatine, to show the numerous small protrusions of synovial membrane through the posterior ligament. (From a specimem in the museum of St. Thomas's Hospital.)

Synovial cysts appear to originate in some cases in the distension of a bursa which normally communicates with the joint, and it is evident that, where there is a direct continuity between the cavity of a joint and that of a bursa, any inflammatory condition may extend from the one to the other.

In other cases the cyst appears to be formed by a hernial protrusion of synovial membrane which has been softened by inflammation and distended by fluid; and in yet other instances it appears that the fluid is free in the tissues and

not confined by any definite sac, the synovial membrane having given way at some point.

In Fig. 99—which is a drawing of a specimen prepared by Mr. Shattock—it is seen very plainly that even in a normal joint there is a tendency to protrusion of small pouches of synovial membrane between the fibres of the capsule, and it is easy to understand that such protrusions might readily increase in case of softening by inflammation or distension by excessive synovial fluid.

Cysts formed in one of these ways may attain considerable size, and I have seen one in connection with the knee which contained a pint of clear synovial fluid.

One of the most important features presented by these cysts is the distance at which the main swelling may be situated from the articulation with which it is really connected. I have seen a cyst which was situated below the middle of the calf, and which did not seem to have any connection at all with the knee, for the latter was freely movable, painless, not swollen, and no fluid could be pressed from the cyst into the synovial cavity. Yet a year and a half later, when amputation of the thigh had to be performed for destructive disease of the joint, I was able to find a narrow track leading from the cyst into the outer side of the knee. The specimen is figured in the accompanying drawing (see Fig. 100). It is evident that such pathological facts as these have a great clinical

FIG. 100.

A Knee-joint with a Synovial Cyst. One piece of catheter has been passed from the articulation into the upper part of the cyst, and another piece along an extension of the cyst on the inner side of the calf.

importance, for, if not recognised, synovial cysts may be mistaken for chronic abscesses, and laid open, with the serious risk of exciting acute arthritis. Synovial cysts may occur in connection with almost any joint, but they appear most frequently in connection with the knee, and, after it, with the hip, elbow, and shoulder.

CHAPTER XLII.

DISEASES OF JOINTS—(*continued*).

TUBERCULAR OR SCROFULOUS DISEASE OF JOINTS.

The general pathology of scrofula or struma has been discussed in an earlier part of this work, and the relation which it bears to tubercle has already been pointed out.

Tubercular disease of joints displays in a most characteristic manner all the essential features of a typical strumous inflammation. The process is exceedingly chronic; is liable to extend from its original seat to neighbouring structures; is attended by little or no tendency to repair; the inflammatory products are very liable to caseate, and chronic abscesses are common.

Joint disease of this nature has been described by various surgeons under different names. Thus, in England, the term "pulpy degeneration of the synovial membrane," originally employed by Brodie, has for long been in common use, whilst, in Germany "tumor albus," or white swelling, has been employed to denote the usual absence of acute inflammatory symptoms.

The patients who are most liable to this form of arthritis are certainly young children from three to twelve years of age, and as puberty is approached, not only does tubercular disease become less frequent, but the tendency to repair is also greater. It would, however, be a grave error to suppose that adults are exempt, for there is no period of life, even up to extreme old age, in which tubercular arthritis may not occur.

The joints most often affected are the hip and the knee; after these the elbow, ankle, and shoulder. Disease of the wrist is not very common in young children, but is comparatively frequently seen in young adults. In some cases there is clear evidence that the arthritis has been started—as may any other strumous process—by an injury; but it is quite certain that this is not always the case, and I have seen tubercular arthritis originate whilst the patient has been at complete rest in bed for the treatment of other affections. Many cases are probably the result of infection from a tubercular focus in some other part of the body.

It is tolerably certain that tubercular disease commences either in the articular bone or in the synovial membrane, and that it is never primary in either the cartilages or ligaments. In some joints, *e.g.*, the knee, the synovial membrane is the starting-place in the large majority of cases; in other joints, *e.g.*, the hip, the bone appears to be primarily affected in most instances.

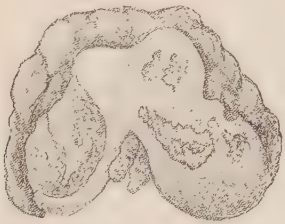
Tubercular Synovitis.—Three varieties of this have been described, of which the first is infinitely the most common, and the third extremely rare, namely (1) "Diffuse Tubercular Inflammation"; (2) "Nodular Tubercle of the Synovial Membrane"; (3) "Miliary Tubercle of the Synovial Membrane."

Taking the knee as a good example of a hinge-joint, and as one in which the disease is often primarily synovial, the following changes may be described as occurring in the first variety, or "diffuse tubercular inflammation."

The synovial membrane becomes swollen, soft, and succulent. Its colour changes to a greyish tint, and, on section, it appears gelatinous. The surface remains smooth for some time, but gradually assumes a roughened or shreddy appearance, and in time becomes converted into true granulation tissue. As the disease advances, the membrane becomes soft, pulpy, and friable, and here and there minute masses of caseous matter develop. The synovial secretion is very slightly increased, and there is

seldom more than an ounce or so of fluid in the synovial cavity. The fluid is, however, altered in quality, being opalescent from admixture with the products of inflammation, and for the same reason containing shreds of fibrin which tend to be deposited on the synovial membrane and cause in part the roughened surface of the latter. The general swelling of the articulation in such cases is, therefore, not the result of effusion into the joint, but of the swollen state of the synovial membrane itself; and it is to the same cause that we must attribute the obliteration of the fossæ on each side of the patella, and the concealment of the normal bony prominences.

FIG. 101.



Ulceration of the Cartilages of the Lower Articular Surface of the Femur.

When fully established in the synovial membrane, the disease soons extends to the **cartilages**. In the earlier stages the latter are simply overlapped at their margins by the swollen membrane, which can easily be lifted off; but, as the inflammatory process extends, the synovial membrane becomes adherent to the margins of the cartilages, and cannot be separated without tearing its structure. If this be done, it will be found that the subjacent cartilage is pitted and ulcerated. The inflammatory process has extended from the soft parts, the cartilage has been vascularised by offshoots of the synovial vessels, and in it the same slow but persistent destructive changes have commenced.

By a continuance of these changes the whole depth of the cartilage is ulcerated through, and the **bone** in its turn is affected. Osteitis supervenes, and the femur, patella, or tibia, as the case may be, becomes the seat of tubercular caries, with its accompanying rarefaction and destruction of the cancellous bone, its caseation of inflammatory products, and its absence of formation of new bone from the periosteum. A clinical examination of a strumous knee-joint often gives the impression that the bones are thickened, and in former times it was customary to speak of "expansion" of the articular ends. It has already been pointed out, at p. 303, that inflamed bone never truly expands, and it may be added that in cases such as those under consideration the feeling of enlargement of the bone is almost always delusive, and that an examination of the parts after removal shows that there is usually no formation of sub-periosteal new bone, but merely thickening and inflammation of the superjacent soft tissues. The **epiphysial cartilages** may be similarly involved, although they are much more likely to suffer when the disease is primarily situated in the bone. In some cases they are destroyed by the tubercular infiltration, and in others they become calcified in parts so that the development of bone from them ceases, and the growth of the affected limb is arrested.

The **ligaments** share in the general destruction. Inflammatory exudation separates the fibres, and destroys their normally dense structure. They soften, become shreddy, and gradually stretch, or ulcerate away.

The whole of the changes described above may occur without the formation of any collection of pus, but this is not usually the case. Inflammatory processes extend to the neighbouring soft tissues, and chronic abscesses either originate in them, or else the softened synovial membrane yields an exit to some of its secretion, and the abscess in the soft tissue directly communicates with the synovial cavity, or with some portion of it which has been shut off by adhesions. When these abscesses burst, and their

contents are exposed to contamination by the external surroundings, the formation of pus in them is often greatly increased on account of a more acute inflammation of the abscess-sac. In some cases this profuse formation of pus subsides after a few weeks; in others, it continues for many months.

A microscopical examination of the tissues shows that the process is of a tubercular nature.

The synovial membrane is infiltrated with inflammatory products, which tend to undergo caseous degeneration and to form small collections of pus. The endothelial lining of the synovial membrane is destroyed, and the interstitial granulation tissue comes to the surface, and discharges its secretion into the synovial cavity. In the midst of this inflamed tissue are numerous primitive tubercles, with their giant-cells and lymphoid reticulum. Tubercle bacilli are also to be found in some cases, but in my own experience they are met with only in a very small number.

The ligaments are infiltrated with cells, by which their structure is slowly destroyed.

The cartilages are vascularised by ingrowths from the contiguous synovial membrane, their matrix is broken up and eroded by the exuded leucocytes, and the cartilage-cells themselves multiply and undergo fatty changes. The alterations in the osseous tissues are similar to those already described as occurring in strumous osteitis.

Nodular tubercle of synovial membrane.—Here the clinical symptoms are rather those of chronic hydroarthrosis than of tubercular arthritis, and the affected articulation may be almost painless and freely movable. I have seen a case of this kind which closely simulated "Charcot's disease." In these cases the whole membrane is much thickened, and its surface presents numerous sessile or pedunculated outgrowths, which are much larger and more flattened than the fringes of osteoarthritis, though some of them may be small and nodular. Their surfaces are smooth, and they do not become ad-

herent to the adjacent cartilages. They are often coated with fibrin, and may give rise to the growth of "loose bodies" in the joint (see p. 381). Examples of this variety of disease are rare, and there may be bone-tubercle as well. The cases run a slow clinical course.

Miliary tubercle of synovial membrane.—Scattered miliary tubercle is very rarely seen on the synovial membrane, except as a complication of other tubercular joint lesions, although it may occur as part of a general tuber-

FIG. 102.

The under surface of the Patella showing the lumpy condition of the synovial membrane in a case of "nodular tubercle."

culosis. These tubercles present the ordinary appearance of grey tubercle, and the joint is usually the seat of a considerable effusion.

Tubercular disease of articular bone.—It has already been mentioned on page 307 that there are three varieties of tubercular osteitis, and any one of these may commence in the articular cancellous bone and extend to the cartilage of the joint. The most common lesion as a cause of tubercular arthritis is the "circumscribed nodule," but "diffuse infiltration" of tubercle may also be the cause of the joint affection. "Tubercular necrosis" is a rare

variety, if we limit the term to the death of considerable portions of bone, and do not apply it to the small dead crumbs which may be shed in any case of tubercular osteitis, but it affects particularly articular bone, and seems to occur more often in the lower end of the femur than elsewhere. These tubercular sequestra are commonly wedge-shaped, the base of the wedge corresponding to the articular surface, and sometimes measuring an inch or more in diameter. One of the most remarkable features of such a case is the complete absence of any bony thick-

FIG. 103.

Lower end of Femur showing tubercular necrosis of each condyle.

ening, or sclerosis around the sequestrum, as in cases of necrosis from other causes, so that usually the outline of the affected bone is not altered, and the condition is scarcely to be recognised until the bone is cut open and its articular surface exposed. Another peculiarity is the very slight tendency to separation of the dead bone, which may indeed maintain its relations to the surrounding osseous tissue for many years. The dead bone, when cancellous, is found to have all its spaces filled up by caseous material, fibrous tissue, and a little new bone. It is probable that the necrosis is the direct result of tubercular osteitis and occlusion of the blood-supply by the disease of the small arterioles.

The position of the diseased joint is always one of flexion, and many theories have been originated to account for the fact. It may be stated at once that the position of flexion is not limited to the knee, but that all diseased joints are liable to be flexed. The most simple explanation, and I believe the only true one, is that it is the normal position of "rest," and is the one in which there is least pain. A moment's reflection is sufficient to convince any one that even in a state of health, flexion is the natural position of rest, and that it is the one in which all textures are most relaxed—in which there is the least tension.

A different explanation is, however, given by some writers. It is stated that when the terminal filaments of the articular nerves are irritated, there is a general tendency to reflex muscular contraction, and that those muscles which are the strongest prevail: the supposed greater strength of the flexors of the knee is therefore held to account for its bent position.

Other surgeons have experimented by injecting fluid into the capsule of the knee or the hip of a dissected limb, and have shown that when the capsule is distended the joint in question is mechanically flexed. This also has been adduced as an explanation of the flexed position of a diseased articulation, but is evidently insufficient. In tubercular cases there is practically never acute distension of the capsule, and, on the other hand, in some cases of great effusion—*e.g.*, in hydrarthrosis—there is no flexion at all. There is indeed no sufficient reason for believing that in strumous disease distension of the capsule ever *mechanically* causes flexion, but I have no doubt that if effusion is present it induces flexion indirectly, for, as I have already said, the joint is *reflexly* placed in the position of rest and of least tension.

As the ligaments yield and the bones ulcerate away, the tibia becomes displaced backwards, partly by its own weight and partly by the continued action of the ham-strings. Further, by reason of the tendency that the lower extremity

always has to rotate outwards if the continuity of its bony supports is in any way interfered with, and perhaps on account of the contraction of the strong biceps muscle, external rotation commonly ensues, and finally the articular surface of the tibia is to a great extent displaced from that of the femur.

The muscles of the thigh and leg are always atrophied in cases of tubercular disease of the knee, and on account of this atrophy, which affects their length as well as their thickness, it becomes impossible after a time to replace the dislocated bones in their normal position. This difficulty is enhanced by the fact that after some length of time the other soft tissues besides the muscles accommodate themselves to the altered position of the parts, and become proportionately shortened on the side of flexion. The tibia and femur in cases of long-standing disease also waste, and cease to develop naturally—a condition which is of much clinical importance in considering the advisability of performing the operation of excision on such a joint.

HIP DISEASE.

The term "hip disease" is used in a general way to imply strumous disease of that articulation, and it is better not to apply it to cases of simple synovitis or acute suppurative arthritis. The process may originate in the synovial membrane, and in that case does not materially differ in its general characters from similar disease above described as attacking the knee.

Hip disease, however, commonly commences in the bones, and originates as a chronic strumous osteitis.

In the femur the disease commences in one of the following localities—(*a*) the cancellous tissue immediately beneath the articular cartilage; (*b*) the centre of the head in the immediate vicinity of the centre of ossification; (*c*) the new bone at the margins of the epiphysial cartilage between the head and neck; or (*d*) in some other part of the neck within the capsule. In the acetabulum the inflammation attacks

first the most recently formed bone in the neighbourhood of the Y-shaped cartilage.

In whatever part of the bones the osteitis commences, it gradually extends until it reaches the surface. Thus, it may extend laterally through the neck, and, by separating the head of the bone from the cartilage of the epiphysis, may cause it to be cast loose into the articular cavity as a sequestrum, or, commencing in the neck, may pass outward until it reaches the periosteum, may penetrate the latter, and thus reach the articular cavity. Beginning, as it most often does, in the cancellous tissue of the head itself, it penetrates to the under-surface of the articular cartilage, and sets up inflammatory processes in the latter, which result either in its perforation by ulceration or in its separation in a necrosed condition. In any case the synovial membrane and ligaments become secondarily affected, and a general strumous arthritis supervenes. As the process continues, the cancellous tissue forming the head of the bone is slowly destroyed, the margins of the acetabular cavity ulcerate away, and the acetabulum itself becomes carious. The head of the bone may now sink into, and penetrate, the carious floor of the acetabulum, or, as is more usually the case, may be gradually displaced upwards over the broken-down acetabular margin on to the dorsum ilii, a change of position often accompanied by a corresponding extension of the carious process to the compact bone of the ilium itself. In other and rare cases, this

Fig. 104.

Portion of a Femur from a case of Hip Disease. The cartilage of the head has been destroyed; the bone is rough and carious, and the epiphysis of the head has become separated. The shaft is roughened by the formation of periosteal new bone.

displacement of the femur is the result of a separation of the head of the bone, the neck, no longer catching in the acetabulum, being readily displaced by the contraction and tension of its attached muscles.

The formation of abscesses is of common occurrence in cases of advanced hip disease. If the pus is first formed within the joint, it makes its exit either at the cotyloid notch, at the thin posterior portion of the capsule, or else into the bursa beneath the psoas muscle. Such abscesses point most often on the upper part of Scarpa's triangle, but not infrequently they pass to the outer side of the limb, and come to the surface a little below the trochanter. In other instances, especially when the ilium is much diseased and the acetabulum perforated, an iliac abscess forms, and points above Poupart's ligament. More rarely, the pus makes its way into the cavity of the pelvis, and opens into the rectum or bladder; or, when the shaft of the femur is affected, extends to a considerable distance down the thigh.

The natural **position** of the limb is much altered in hip disease, and that in a very characteristic manner. In the early stages the thigh is flexed, abducted, and rotated outwards. This position is the one which gives the patient most ease, and is the natural position of the lower extremity when at rest. Flexion relaxes the ilio-femoral ligament; abduction, the ligamentum teres; and rotation outwards, the inner portion of the capsule. In the later stages, when the ligamentum teres has been softened or destroyed and the capsular and other ligaments are stretched, the rotation outwards and adduction are replaced by rotation inwards and adduction, for in the relaxed state of the ligaments the latter position tends to relieve pain by displacing the head of the bone from the floor of the acetabulum, and so preventing pressure between the opposed carious surfaces.

In consequence of the altered relations between the femur and the innominate bone, and the fixation of the hip-

joint by the muscles, the patient is led to the adoption of certain positions in order to enable him to put the foot to the ground. The hip-joint being fixed in a state of flexion, the mobility of the lumbar spine is brought into play, and the flexed limb is placed in a line with its fellow by rotating the pelvis on its transverse axis and arching forward the lumbar spine (lordosis). If the femur is abducted, the pelvis is tilted downwards, so that the anterior superior spine of the diseased side is placed on a lower level than is that of the opposite side, and the limb, when the patient is placed in a recumbent posture, is "apparently lengthened." If, on the other hand, the femur be adducted, the pelvis is tilted upwards until the thigh is placed parallel with its fellow ; the iliac spine is thus placed on a higher level than is that of the sound side, and "apparent shortening" results.

Real lengthening of the limb in hip disease probably never occurs, but real shortening is produced either by absorption of bone, or by disease of the epiphysial cartilage and consequent arrest of growth. It is important to remember that, after all active disease has subsided, a limb may grow more slowly than that of the healthy side, and that, when a child has recovered without any shortening, it does not follow that in the future the limb will be as long as its fellow.

In any case of tubercular joint disease, a natural cure may result, the limb either completely recovering or being fixed by anchylosis (see p. 403). In many instances, however, in which the disease has not been treated in any early stage, the patient loses his life. A fatal termination may be brought about in one of several ways.

In some patients, death results from exhaustion, arising from the constant pain and want of rest, combined with the suppurative or hectic fever which accompanies the formation of large quantities of pus. In others, amyloid disease of the liver, kidneys, and intestines supervenes, and in the remainder acute tuberculosis or tubercular phthisis terminates the case.

The fact that tubercle is present in the joints in the majority of cases of so-called strumous disease is naturally of great clinical importance, and affords a ready explanation for the occasional development of a more general tuberculosis. Many surgeons, acting upon the assumption that the danger of general infection is considerable, have therefore advised the early excision of all strumous joints. There can, however, be no doubt that treatment founded on such ideas is not supported by pathological investigations, and it is quite certain that the dangers of general tuberculosis in any case of early joint disease placed under proper treatment are very slight. On the other hand, it is now the opinion of various Continental surgeons that operative interference with tubercular joints is itself liable to promote general infection, and I have myself seen several cases in which excision has been followed by the rapid formation and diffusion of tubercle. Lastly, it should be remembered that, in even the most complete excisions, it is generally impossible to be certain of removing all the tuberculous tissues.

SYPHILITIC DISEASES OF THE JOINTS.

Syphilis may affect the joints in one of three ways—(1) In the early periods of the constitutional affection, at the time when the patient is suffering from the so-called secondary symptoms, one or more articulations may be the seat of a subacute synovitis. There are no records of post-mortem examinations of such joints, but it is probable that the changes in them do not differ from those met with in cases of simple serous synovitis.

(2) In the latter stages of syphilis, at a time when the patient is subject to gummatous or rupial ulcers, to necrosis or caries of the bones, &c., the synovial membrane may be the seat of inflammatory effusion, and gummatous masses may develop in the synovial and sub-synovial tissues. In such cases there is much thickening of the affected parts,

and considerable destruction of the articular structures. (See Fig. 90.)

(3) The articular extremities of the bones may be affected in a manner similar to the shafts, and an inflammation, commencing in the periosteum, may extend to the neighbouring joint.

Considering the prevalence of syphilis, the joints are comparatively rarely affected in this disease.

In **inherited syphilis** also the joints may be affected in the same manner as in the last two varieties just

FIG. 105.

Knee-joint from a Man who died of Syphilis. The synovial membrane is thickened and infiltrated by gummatous deposit.

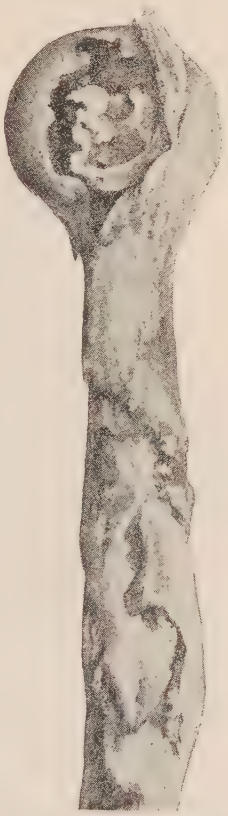
mentioned, but, in addition, there are special varieties of arthritis which are peculiar to the inherited form of the disease, and apart from the lesions about the epiphysial cartilage, which have already been described in the chapter on "congenital syphilis."

First, there is a form of very chronic synovitis which occurs usually between the ages of eight and fifteen, and is most common about the middle of this period. It affects especially the knees, is of gradual onset, and results in very considerable effusion without any severe pain. The patient is seldom incapacitated, and flexion and extension movements are generally smooth and free. The condition of

the joint is most often one of hydro-arthrosis, and the articular bone does not show any signs of disease. Although these cases are not very rare, there do not appear to be any recorded examinations of joints thus affected.

Second, the articular cartilage and bone, as well as the synovial membrane, may be the seat of very peculiar lesions, which appear to have been first noticed by Virchow, and of which there are only three or four recorded cases. A series of such joints, all from the same patient, has recently been added to the Museum of St. Bartholomew's Hospital, and the case has been recorded in vol. lxx. of the Royal Medical and Chirurgical Society's "Transactions." The cartilage is affected in two ways. There is overgrowth and the development of ecchondroses, giving the joint very much the appearance of one affected by osteo-arthritis; and there is also a peculiar gouging out of the articular cartilage in many places, resulting in crescentic or circular cavities in it, some of which penetrate its whole depth and involve the subjacent bone. The latter is rarefied at such points and may be destroyed to a depth of a quarter of an inch or more by a chronic osteitis which leaves its articular surface pitted and deformed. The synovial membrane in the specimen in

FIG. 106.



Disease of the Humerus from a case of Congenital Syphilis, showing the gouging of the articular cartilage, and the ulceration of the compact bone.

St. Bartholomew's Museum is most extensively altered, being greatly thickened, and presenting over its entire surface large pendulous fringes, giving it a shaggy appearance, and simulating very closely the fringed membrane of osteo-arthritis. There are no gummata in the synovial membrane and it does not adhere to the neighbouring

FIG. 107.

Disease of Knee from a case of Congenital Syphilis, showing the fringed synovial membrane and the gouging of the articular cartilage of the femur.

cartilages or ligaments. The synovial fluid is greatly increased in quantity. In the case to which allusion has been made the disease was very chronic, and was characterised by swelling of the joints with some stiffness and pain on movement.

ANCHYLOSIS.

The term anchylosis is commonly employed to indicate a condition of complete, or partial, immobility of a joint due to adhesions between different portions of the articular surfaces.

In all cases anchylosis is the result of pre-existing

inflammation, and the extent, position, and density of the adhesions depend upon the tissues which have been involved in the inflammation, on the amount of destruction which the tissues themselves have undergone, and on the nature of the inflammatory process. Where the inflammation has been acute, and has progressed to the formation of pus in large quantities, as in acute suppurative arthritis, the anchylosis is liable to be very firm and bony, whilst in cases of simple chronic synovitis there is usually no attempt at the formation of any adhesions. In tubercular disease, where there is little tendency to repair, anchylosis is liable to be not very firm, whilst in osteo-arthritis anchylosis never results except in the spinal joints.

Fig. 108.

An Anchylosed Knee-joint. The patella is fixed to the femur by bone, but the rest of the anchylosis is by fibrous tissue. The tibia has been dislocated backwards to a considerable extent.

In those cases where the inflammatory processes have involved the synovial membrane, the ligaments, and the cartilages only, the adhesions are composed of fibrous tissue; but where the bones have been affected, and especially when there has been suppuration, bony anchylosis or osseous union of two granulating surfaces is common.

Adhesions may be formed in the following ways :—

(1) If two layers of synovial membrane in a state of inflammation are kept in constant apposition, their surfaces may become united by fibrous tissue, just as may the opposed surfaces of the pleuræ. Thus, in the knee-joint, it is common to find the anterior and posterior surfaces of the synovial pouch beneath the quadriceps tendon adherent, and the pouch in many cases obliterated.

(2) Two opposed surfaces of ulcerating cartilage may be

united by the development of fibrous tissue between them—*e.g.*, the patella may be fixed to the external condyle of the femur.

(3) Inflammatory exudation into the ligaments may become developed into fibrous tissue, and the latter by its subsequent contraction, may interfere with the proper movements of the articulation. Thus, in disease of the knee, when the joint has long been kept in a state of flexion, the posterior ligament of Winslow may be found permanently thickened and contracted, and may frustrate all attempts at complete extension.

FIG. 109.

Hip-joint showing Complete Bony Anchylosis.

(4) Two opposed osseous surfaces in a state of inflammation may join to one another by bone in the same manner as the ends of a bone unite after a fracture. Sometimes this union is so complete that the two bones are welded together in such a manner that their line of junction cannot be distinguished. To such a condition the name of "synostosis" has been applied.

In addition to these varieties of anchylosis, it must be remembered that spurious anchylosis, or fixation of a joint by changes in the tissues outside it, is of common occurrence. This may be brought about in various ways.

(*a*) Where a joint is diseased, the muscles which regulate its movements cause partial or complete immobility by tonic reflex contraction, and thus prevent the patient from suffering the pain which would result from friction between the articular surfaces.

(*b*) Inflammatory exudation around a joint may cause thickening and adhesion of the peri-articular tissues, such as the tendons, muscles, or fasciæ.

(*c*) In cases where a joint has been long maintained in a bent position, the soft tissues on the side of flexion accommodate themselves to the altered position of the bones, and finally become too short to allow of complete extension. This condition may often be seen in cases of long-standing disease of the knee-joint, and is liable to cause much difficulty in cases of excision.

CHAPTER XLIII.

DISEASES OF THE PENIS AND SCROTUM.

ECTOPIA VESICÆ AND EPISPADIAS.

Ectopia vesicæ, or extroversion of the bladder, consists of a deficiency of the anterior vesical wall and of the corresponding part of the abdominal parietes, and results from failure in closure of the lower part of the fœtal body-cavity.

In such cases, the bladder, being incomplete in front, fails to act as a reservoir, and the urine escapes as rapidly as it passes from the ureters. The posterior vesical wall is thrust forward, just above the pubes, by the pressure of the abdominal viscera, and presents a mucous surface, which is often swollen and inflamed, either on a level with the surrounding skin or else protruding in front of it. On this surface the orifices of the ureters may be seen, the urine being passed from them at short intervals in minute jets. The urethral orifice is always imperfect, and the penis itself is small and in a condition of "**epispadias**," the corpora cavernosa being cleft and the roof of the urethra undeveloped, so that the urine flows along a shallow groove instead of through a mucous canal. The pubic bones are usually separated in the middle line by a considerable interval, the recti muscles consequently diverging to reach their attachments. The testes are commonly retained, and inguinal herniæ are of frequent occurrence. The deficiency in the abdominal wall extends as high as the umbilicus.

HYPOSPADIAS.

Hypospadias is the term applied to a cleft condition of the floor of the urethra, which then forms a groove instead of a canal on the under surface of the penis. It results from imperfect fusion of the two sides of the uro-genital aperture, which in early fœtal life forms an antero-posterior slit or fissure at the lowest part of the abdomen.

In the female, this fissure does not close, and, while its two lateral boundaries form the labia, the clitoris is developed at its anterior commissure. In the male, the two halves normally unite, forming the scrotum below, and closing in the floor of the urethra anteriorly.

In slight forms of hypospadias, the glans and prepuce alone are cleft. In more severe cases the urethra is opened up as far back as the scrotum, whilst in complete hypospadias the fissure extends as far back as the membranous urethra, the scrotum being divided in the middle line. In such cases as these the cleft scrotum, with its contained testes, closely resembles the labia, and the penis, being very imperfectly developed, aids the delusion. It is by no means uncommon for male children with this deformity to be brought up as females, the error not being discovered until the period of puberty approaches. When hypospadias is slight, it causes no inconvenience, but in those cases where the whole urethra is affected the penis during erection is curved downwards, and connection is rendered impossible.

PHIMOSIS.

The term phimosis is employed to indicate any condition of the prepuce which prevents its retraction over the glans penis. In the great majority of cases phimosis is a congenital defect, and one which often gives rise to troublesome symptoms. It causes retention of the secretion of the sebaceous glands in the neighbourhood of the corona glandis, and consequent continued irritation from the presence of the retained matter. If the condition is not

relieved, the under surface of the prepuce is liable to become adherent to the glans. Vesical irritability, with frequent micturition, straining, herniæ, prolapse of the rectum, and noctural incontinence of urine are amongst the most frequent troubles which arise from this affection.

Phimosis may also result from inflammatory swelling in connection with gonorrhœa or venereal sores, but is then usually transient. In some cases, however, the contraction of a cicatrix causes permanent narrowing of the preputial orifice, and in old men the same condition is liable to supervene upon the cracks and fissures which are not infrequently met with on the prepuce.

PARAPHIMOSIS.

Paraphimosis results from retraction of a tight prepuce behind the corona glandis, where it remains fixed. Paraphimosis may occur in connection with a congenitally tight preputial orifice, but it more often follows upon retraction of an inflamed prepuce in a case of gonorrhœa. In consequence of the constriction of the glans by the tight preputial orifice, it becomes congested, whilst at the same time the constriction also tightens and the prepuce becomes swollen and œdematous. If the condition be not relieved, the tight preputial orifice usually ulcerates or sloughs in some part of its circumference, but the glans penis itself hardly ever becomes gangrenous.

EPITHELIOMA OF THE PENIS.

Epithelioma of the penis, as of other parts, occurs chiefly in old men. It almost invariably commences on the prepuce or the glans, and is very rarely seen as a primary affection of the body of the penis. Phimosis, or cracks and fissures of the prepuce, are generally believed to act as predisposing causes.

Commencing as a warty growth, the increase of the epithelioma is generally rapid. Its surface soon ulcerates, and the deeper tissues become at an early stage infiltrated

and indurated. If left alone, it extends along the corpora cavernosa and the corpus spongiosum to the perineum, and subsequently to the neck of the bladder. Obstruction to the outflow of urine, with severe cystitis, supervenes, and life is often terminated by kidney disease resulting from the foul and unhealty state of the bladder. The inguinal glands are early enlarged by formations in them of secondary growths, and in many cases similar disease extends to the deeper lymphatic glands along the iliac vessels and in the lumbar regions. Deposits may also form in the viscera, but are not of common occurrence.

Innocent tumours of the penis are rare, and do not require special mention. Venereal warts are treated of in the chapter on Gonorrhœa.

CHIMNEY-SWEEP'S CANCER—EPITHELIOMA OF THE SCROTUM.

The occurrence of epithelioma of the scrotum is undoubtedly caused by the habitual presence of soot in its rugæ, and it may be mentioned that, in sweeps, epitheliomata appear specially liable to develop on other parts of the body. It is commonly stated, and believed, that epithelioma of the scrotum is becoming less common, but it is doubtful whether this is really the case.

The disease commonly commences as a wart, which, after a time, ulcerates and extends, as do epitheliomata in other parts. Even when apparently a simple wart, the disease is really of a malignant nature, and I have had opportunities of verifying this statement by microscoipcal examination of growths no larger than a split pea. If untreated, the growth extends to the perineum and penis, in much the same manner as when epithelioma commences in the latter organ. The inguinal glands are also early occupied by secondary growths which may also become disseminated in the viscera.

Innocent tumours of the scrotum are comparatively rare, and of no special importance. The most common of them is the soft fibroma, which occasionally attains a considerable size.

CHAPTER XLIV.

GONORRHŒA AND STRICTURE.

GONORRHŒA.

Gonorrhœa is a specific inflammation of the urethra and contiguous parts, and is always transmitted by contagion. A micrococcus, to which the name of gonococcus has been given, has been found to be constantly present in all cases of gonorrhœa; the organism in question occurs typically in pairs or in fours and differ from similar cocci in refusing to stain by Gram's method. Before entering into a description of gonorrhœa, however, it may be pointed out that all forms of urethritis are certainly not of gonorrhœal origin, many of them being due to infection by irritating secretions from the vagina or uterus, or to the passage of instruments. In other cases the presence of uric acid crystals in the urine may cause urethritis, and in gouty subjects this may be accompanied by a profuse purulent discharge.

In gonorrhœa the inflammation, originated by the application of gonorrhœal pus to the glans penis and orifice of the urethra, often extends along the whole length of that passage, though it is usually most intense in the fossa navicularis. The mucous membrane is swollen and intensely injected, and a secretion, at first of mucus and serum, but very soon of pus, is rapidly established. The amount of pus discharged is usually very great, and in some cases the distended capillaries give way and allow a discharge of blood. After lasting for a week or more, the acute inflammation slowly subsides, the discharge becomes less abundant and less purulent, and, after persisting as a watery exudation for a variable time, finally entirely ceases.

During the height of the attack, the corpus spongiosum is itself infiltrated with plastic effusion, and the whole penis is swollen and tender. There is usually much pain, and increased frequency of micturition, often accompanied by a good deal of fever. The swelling of the urethral mucous membrane frequently causes some difficulty of micturition, and occasionally induces complete retention of urine.

FIG. 110.

(1) Gonococci. (2) Gonococci in pus-cells. (3) Gonococci in an epithelial cell. From Flügge's "Hygiene."

Painful erections of the penis, or "chordee," are an almost constant accompaniment of the acute stage, and are characterised by distortion of the organ so that it forms a curve, with the concavity downwards. This condition results from the presence of plastic exudation in the corpus spongiosum, and a consequent interference with its erectile tissue.

COMPLICATIONS OF GONORRHŒA.

The complications of gonorrhœa are so numerous that it is impossible to describe all of them at length, but they are of sufficient importance to require some mention.

As regards the **penis** itself, either phimosis or paraphimosis may arise, as already described, and inflammation of the prepuce (posthitis) and of the glans penis (balanitis) are of frequent occurrence. Inflammation of the lymphatics of the penis, and localised inflammatory induration of the corpus spongiosum or corpora cavernosa, are more rare. Warty growths about the prepuce and glans generally

result from want of cleanliness in cases where the discharge has lasted a long time.

Inflammation of the lymphatic glands in the groin is common, and is very liable to terminate in suppuration or the formation of a "bubo." The pus formed in the glands under these circumstances does not as a rule contain gonococci, and the ulcers are consequently unhealthy and little prone to heal. In rare cases the glands in the iliac fossa are affected, and deep-seated suppuration ensues.

Epididymitis and **orchitis** are amongst the most common of the complications of gonorrhœa, and are more prone to develop in the second or third week than at an earlier date. They probably result from a direct extension of the inflammation from the prostatic urethra to the ejaculatory ducts and the vas deferens, but are considered by some authors to be of metastatic origin. In many cases the onset of the epididymitis is followed by a diminution of the urethral discharge, and its subsidence by a return of the urethritis. For further details the chapter on the Testes may be consulted.

Urethral or peri-urethral abscess is to be placed among the rarer complications of gonorrhœa. It may develop along any part of the corpus spongiosum, but is most common in the perineum and close to the urethral orifice. Such abscesses are supposed to result from suppuration around one of the lacunæ in the urethral floor. If opened early, they do not usually communicate with the urethra, but, if left alone, they are liable to open into the latter.

Acute and chronic **prostatitis** are fortunately comparatively infrequent. They are more fully described in the chapter on the Prostate.

Cystitis in its milder form is not uncommon, whilst acute cystitis, though rare, is a most serious and dangerous complication. Exceptional cases have been recorded of extension of inflammation along the ureters to the kidneys, with consequent pyelitis and nephritis.

Gonorrhœal ophthalmia is one of the most acute

and serious forms of inflammation to which the eye is subject. Commencing in the conjunctiva, the inflammation rapidly extends to the cornea and sclerotic, is accompanied by much purulent discharge, and is liable to terminate in sloughing or ulceration of the cornea, and consequent destruction of the entire globe. It is most probable that gonorrhœal ophthalmia and many cases of "ophthalmia neonatorum" are always the result of contagion by gonorrhœal pus, and it is evident that the other eye is very prone to be implicated by inoculation with the discharge of its fellow.

Gonorrhœal **sclerotitis** is usually associated with gonorrhœal rheumatism, and, beyond being exceedingly intractable and chronic, does not differ from sclerotitis arising from other causes.

Gonorrhœal rheumatism has already been dealt with in a previous chapter.

Gonorrhœa in women is rather an inflammation of the vagina than of the urethra, although the latter is also frequently implicated. Its complications are much the same as in the male, but differences arise, of course, in connection with the different parts involved.

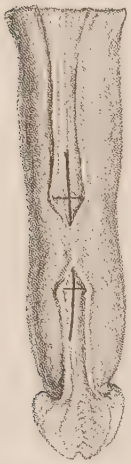
Inflammatory œdema of the vulva is sometimes very marked, and the swelling great. Vulvar abscesses are also common. In rare cases the cavity of the uterus is involved in the inflammation, which may also extend to the Fallopian tubes. Ovaritis is probably more common than is generally supposed, and peritonitis occasionally results from extension of inflammation of the ovaries or the tubes. Chronic suppuration of the tubes, with distension by pus, or "pyosalpinx," and consequent formation of adhesions to surrounding parts, is usually considered to result from gonorrhœa more frequently than from any other cause.

STRICTURE OF THE URETHRA.

Stricture of the urethra, or narrowing of its calibre, is commonly described as of three varieties—(1) congestive; (2) spasmodic; (3) organic. In the present chapter, it is

to the last of these varieties alone—the organic—that the term will be applied. Congestive stricture is but another name for inflammatory swelling of the mucous membrane, such as has already been described as occasionally causing retention of urine in gonorrhœa. Spasmodic stricture does not exist *per se*, though spasm may complicate either an organic stricture or any irritated or inflammatory condition of the bladder or urethra.

FIG. 111.



A Urethra laid open showing a stricture about two inches from the meatus.

Organic stricture is a narrowing of the urethra by the formation of fibrous tissue, and results almost invariably from gonorrhœa, chancre, or injury. It has already been said that, after the acute inflammatory stage of gonorrhœa has subsided, a chronic urethritis, or "gleet," is apt to persist for some time. It is to this chronic inflammation of the urethra, rather than to the acute urethritis, that the organic stricture is to be attributed. All chronic inflammations are more liable to cause the formation of fibrous tissue, and consequent induration and thickening, than are acute attacks, and thus the urethra is narrowed by the contraction of scar-tissue formed in its walls.

In cases of laceration, also, the torn urethra is mended by a scar of fibrous tissue, and this, like all other scars, tends to contract, and thus to narrow the tube. These strictures, which are named **traumatic**, are almost always situated in the bulbous or membranous urethra, and are often very tight and not easily dilatable.

Strictures resulting from gonorrhœa may affect almost any part of the urethra. Those seen in museums are most

common in the membranous and bulbous portions, but the less severe varieties, which do not so often find their way into museums, are at least as common, if not more common, in the penile urethra. Strictures at the meatus sometimes result from another cause, namely, the contraction of scars after the healing of venereal sores. The prostatic urethra is never the seat of stricture.

Strictures are sometimes classified according to the amount and arrangement of the fibrous tissue of which they are composed. Thus, when the latter forms a narrow ring round the canal, the stricture is called annular; when it surrounds the tube for a greater portion of its length, tubular; and when bands of fibres pass across the lumen of the tube from one wall to another, the term bridle stricture is employed. A stricture is generally narrower and less dilatable the greater the amount of its fibrous tissue, and to those strictures in which the urethra is buried in a mass of cicatricial tissue the term "cartilaginous" is applied, on account of their great density and hardness.

With regard to the position of a stricture, it may be said that the nearer it is to the meatus the less capable is it of dilatation, and strictures of the meatus itself often yield only to cutting.

EFFECTS OF STRICTURE.

The effects of stricture on the urinary organs are many and serious. They are usually in proportion to the amount of obstruction to the passage of urine, and may be to a great extent prevented by proper treatment. Many slight strictures are never followed by any serious complications.

The **urethra** behind the stricture is in some cases thickened, for its muscular coat hypertrophies in order to overcome the obstruction. More often the tube is dilated by the backward pressure of the urine, and is sometimes much pouched.

The mucous membrane is often in a state of chronic inflammation, and is not infrequently ulcerated and ragged,

the lacunæ being enlarged and forming little pockets in which urine and inflammatory exudation collect. In bad cases the inflammation extends to the tissues around the urethra, and results in the formation of pus in this situation. In other cases ulceration completely penetrates the urethral walls, and a peri-urethral abscess is formed. This abscess in time makes its way to the surface, and bursts, thus establishing a communication with the exterior, and forming a **urinary fistula.** In these cases extravasation of urine is prevented by the plastic effusion into the peri-urethral tissues, but if the thinned and ragged urethra yields suddenly during the act of micturition, or if the walls of the abscess are thin and tear, then extravasation of urine results, the urine being propelled through the rent in the urethral walls, with all the force of an hypertrophied bladder, into the cellular tissue.

The urethra almost always gives way in the membranous or bulbous portions, and the urine, guided by the attachments of the fasciæ, infiltrates the scrotum, penis, and abdominal walls, causing sloughing of the cellular tissue wherever it extends. In bad cases large portions of the urethra itself may slough.

Retention of urine does not result from obliteration of the urethra, but is always brought about by some complication.

It has already been said that the mucous membrane in the neighbourhood of the stricture is often inflamed, and it is easy to understand that anything which causes an increase of this condition—*i.e.*, anything which excites active congestion, may induce such an amount of swelling as to temporarily block up the already obstructed tube. In addition to this, spasm of the muscles at the neck of the bladder or of the urethra itself, may result from the congestion, or from an acid state of the urine. Again, where there is any peri-urethral inflammation or collection of pus, the pressure of the inflammatory products may obstruct the flow of urine. It will thus be seen that the

final cause of retention is almost always some local spasm, congestion, or inflammation, and a knowledge of this is the true key to all efficient treatment. Such conditions are usually transient, and, when they have subsided, the urine is often again voided voluntarily, and instruments which could not previously be passed are readily introduced.

However long urine is retained the bladder hardly ever gives way. In cases of stricture, this viscus is usually hypertrophied, and by its involuntary contractions not only causes the patient much pain, but also over-distends the urethra behind the stricture. It is in consequence of these conditions that in bad cases of retention the urethra bursts, and the urine is extravasated.

FIG. 112.

A Penis with a Stricture of the Urethra in the Membranous Portion. The bladder walls are greatly hypertrophied.

The bladder in all long-standing cases of stricture becomes hypertrophied—a condition which results from the increased force required to expel the urine. The muscular coat of the bladder is normally arranged in interlacing bundles or fasciculi, and as these becomes greatly increased in size in all cases of hypertrophy, and are seen as prominent bands beneath the mucous membrane, the term "**fasiculated**" is often applied to such bladders. On account of this arrangement of the muscular coat, the mucous membrane between the different fasciculi is insufficiently supported, and when the bladder contracts and the tension of the fluid in it is raised, the

mucous coat tends to yield, and to protrude between the bundles of muscular fibres. Such a protrusion is called a "**sacculus**," and a bladder so affected is said to be "**sacculated**." Sacculi vary much in size, and are sometimes sufficiently capacious to hold as much as a pint of urine. However large they may be, their orifice of communication with the bladder is always small. They are liable to cause much trouble, for, having no muscular wall, they are unable to empty their contents satisfactorily, and urine remaining in them is liable to decompose and so to keep up cystitis. In other cases the sacculi contract adhesions to the intestines and other neighbouring structures, and inflammation spreading from them may be the cause of peritonitis.

The thickened bladder is often the seat of **cystitis**, and where this has been of long standing the mucous membrane is much pigmented, and of a dull slate colour. Ulceration is not common. The dilatation of the ureters and disease of the kidneys, which may follow on stricture, are described in the following chapter.

CHAPTER XLV.

SURGICAL DISEASES OF THE KIDNEY.

SURGICAL KIDNEY—PYELO-NEPHRITIS.

The term surgical kidney has long been applied to those cases in which the renal organs become diseased as a result of some obstruction to the outflow of urine from the bladder, but precisely similar pathological conditions may result in a single kidney from any mechanical blocking of its ureter. The name is a bad one, for it seems to indicate that such kidneys result from surgical interference, whereas the reverse is the case; they result from want of proper surgical treatment. Pyelo-nephritis has of late been suggested as an alternative name, but is itself not altogether free from objection.

Causes.—The diseases of the urethra and bladder which cause surgical kidney act in more ways than one. First, it may be said that any persistent irritation of these parts is liable to cause reflexly chronic hyperæmia of the kidneys—a condition which, when prolonged, results in cell exudation and the formation of fibrous tissue between the tubules, and thus causes "interstitial nephritis." Second, anything which obstructs the outflow of urine from the bladder tends to obstruct the flow of urine from the ureters into the bladder. At first sight this does not appear quite clear, for the cavities of the ureters are not directly continuous with that of the bladder, their orifices being valvular and never becoming dilated. Why, then, should they have

difficulty in emptying their contents? The answer is two-fold. First, obstruction to the exit of urine causes hypertrophy and induration of the bladder-walls, and, as the ureters pass obliquely through the thickened tissues, they

FIG. 113.

A Kidney and Ureter. The pelvis and calices are dilated and the renal tissue has been in great part absorbed. The ureter also is much distended. (From a patient who died with a large calculus in his bladder.)

are thus subjected to compression. Second, if the bladder is frequently over-distended, the tension within it is at such times so greatly raised that it is difficult for more urine to find an entrance. In both these ways the urine is retained under pressure in the ureters, and these tubes become first hypertrophied and afterwards distended. But the ureters

are directly continuous with the pelves and calices of the kidneys, and thus these also are distended by the retained urine. This distension of the renal cavities causes chronic irritation of the kidneys, and so interstitial nephritis, as well as absorption of the glandular tissue itself. It is a rule of pathology that constant pressure induces atrophy, and the pressure of the retained urine causes atrophy or absorption of the pyramids, whilst it simultaneously distends the pelvis and calices, and in time reduces the gland to a collection of cysts bounded only by the distended capsule and by thin septa of renal tissue.

Morbid appearances.—Examination of the kidneys and their ureters in these cases shows a variety of conditions, for whereas, in some, inflammatory changes with induration and contraction prevail, in others the effects of distension are most marked.

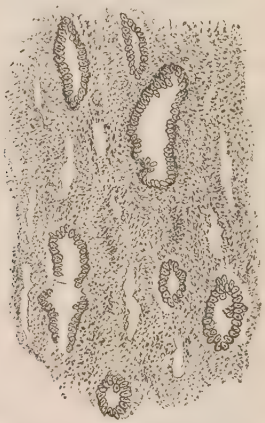
Thus, the **ureters** are thickened both by an increase of their muscular coat and by the formation of fibrous tissue. They may be distended in very varying degrees. It is common to find them as large as a crow-quill, but in exaggerated cases they may attain the size of the œsophagus or of a piece of small intestine. The more they are dilated, the more tortuous and pouched do they become. The longer and the more often they have been inflamed, the darker and more pigmented does their mucous lining appear.

The kidneys are generally found to be unusually adherent to the fat in which they lie. When chronic interstitial nephritis has been more marked than distension of the pelvis and calices, the glands are small; when the latter condition has prevailed, they may be distended to almost any size. The capsules are usually adherent. Small cysts, the size of peas, are found in the cortex, and result from the dilatation of tubules which have been obstructed by the pressure of the surrounding fibrous tissue. On section, the renal tissue is found to be tough and resistant, the cortex is irregular and puckered, and the

renal substance itself is more or less atrophied, as already explained.

On **microscopical examination** the fibrous stroma of the kidney is seen to be increased in quantity, the renal tubules being separated from one another by a growth composed of small cell exudation and of connective tissue of varying density. The new tissue is often very vascular, and in some cases the walls of the vessels are increased in

FIG. 114.



Section through the Cortical Portion of a Surgical Kidney. There is much increase of connective tissue, and the remaining renal tubules are undergoing cystic dilatation. (Zeiss, A.)

thickness. The renal epithelium varies much in different tubes; in some it is natural, in others granular or fatty, whilst some tubules are denuded of their epithelial lining. Distension of the tubules is common, and definite cysts are often formed in the cortex.

ACUTE SUPPURATIVE NEPHRITIS.

A kidney in such a condition as that above described is liable to become at any time the seat of acute inflammation and suppuration, and to pass into a state of "acute suppurative nephritis." Suppurative nephritis usually results

from an extension of inflammation from the bladder, and is often induced by an attack of retention of urine, by frequent or forcible catheterisation, and by operations on the urethra or bladder. The fact is, that the kidneys in such cases are in such a precarious state that a mere trifle may throw them off their balance, and exposure to cold or wet, such as might in normal circumstances induce a slight congestion, will in these cases bring on an acute nephritis. It is for this reason that surgeons are so careful about operating on patients in whom these conditions are suspected, for the most trifling operation, especially on the urinary organs, may induce suppuration in the kidneys, and complete suppression of urine. If a post-mortem examination be made after death from such a cause, the following conditions will usually be found:—

Morbid appearances.—Both kidneys are generally affected, but one is often more diseased than the other. Each is larger than natural, and in some cases is three or four times the normal size. The capsules usually peel off easily, having become loosened by exudation beneath them. The surface of the gland is mottled. In some places it is dark and congested: in others, small collections of pus are seen thinly covered by renal tissue. A section liberates much pus mixed with urine, which usually smells badly. The pelvis and calices are congested, and of a dark-purple or slate colour. The renal tissue is very soft and friable, and parallel with the renal tubes are white streaks of pus, which often lead up to the abscesses in the cortex.

Microscopical examination shows a general infiltration of the kidney with leucocytes. These are in places collected into masses, and represent areas in which the inflammation has progressed to the formation of an abscess. The renal epithelium is swollen, and the tubes are often filled with cast-off cells and leucocytes. Here and there collections of red blood-cells are found, indicating that an over-distended vessel has given way, and its contents have escaped. Micrococci are found in all parts of the section,

especially in the neighbourhood of the developing abscesses.

The pathology of acute suppurative nephritis has been much discussed, and various explanations have been advanced. It appears probable that in most cases the condition is the result of acute inflammation propagated from the bladder to the pelvis and calices, and extending thence to the renal tissue; but in some the inflammation seems to originate in the renal tissue itself, and is then probably the result of acute congestion occurring in an organ already in a state of chronic inflammation.

MOVABLE OR FLOATING KIDNEY.

Movable or floating kidney is the term used to indicate an undue mobility of the organ, which is most commonly met with in women of middle age who have borne children. The kidney most often affected is the right, and the extent of its mobility differs much in different cases. Sometimes it can only be moved an inch or so from its normal situation, whilst in exaggerated cases the gland can be made to travel into all the regions of the abdomen, and I have on post-mortem examination found a kidney so freely movable that I could place it on the promontory of the sacrum and pass it across the spine. In such cases as this the kidney may be found surrounded by a complete peritoneal fold or "mesonephron."

The causes of mobility of the kidney are various. It is possible that the condition is sometimes of congenital origin, and in other cases there is a distinct history of injury. Frequently the mobility is first noticed after pregnancy, and is then attributed to the sudden alteration in the tension of the abdominal contents which ensues upon parturition, as well as to the very flaccid condition of the abdominal walls which occasionally results. In some cases it is probable that the gland may be displaced by tight

lacing, and in others the mobility apparently results from the absorption of renal fat from any cause.

Finally, it is tolerably clear that attacks of hydronephrosis, from whatever cause arising, may produce undue mobility, for, if the pelvis be greatly distended by fluid and then collapses as the urine escapes, it is evident that by this means the surrounding fat and cellular tissue are first displaced, and subsequently become lax when the organ shrinks within them. In consequence of this, movable kidney is often associated with some disease of the uterus or ovaries which causes pressure on or dragging of the ureter.

But, whilst hydronephrosis is thus an occasional cause of floating kidney, in many cases the mobility itself results in a certain amount of hydronephrosis. The mobility of the gland is very liable to cause a bending or kinking of the ureter, and, as a result of this obstruction to the passage of urine, the pelvis and calices may become distended. Not only, however, may the ureter be thus interfered with, but it is also probable that the renal vessels may become twisted through the gland rolling over and causing a twist in its pedicle.

In many cases floating kidneys cause no symptoms whatever, but in others they give rise to a sickening pain when they change their position, and some patients are liable to sudden attacks of severe pain, accompanied by vomiting, which are perhaps the result of a twist in either the vessels or the ureter, or in both.

TUMOURS OF THE KIDNEY.

The new growths of the kidney are mostly malignant, and comprise both carcinomata and sarcomata. Of the former, epithelioma of the pelvis is the most common form, and usually causes profuse hæmaturia; encephaloid cancers also occur, and generally originate in the gland tissue itself.

Sarcoma of the kidney is of comparatively rare occurrence, and may be met with at all ages. The tumours are

generally soft, friable, and very vascular. They attain a considerable size, and are prone to affect the neighbouring glands and to disseminate.

The malignant tumours of the kidney which occur in infancy and childhood have a structure peculiar to themselves, for whilst they consist to some extent of sarcomatous tissue, they are composed in great part of glandular tissue. This is arranged to form tubes and spherical cavities, or cysts, which are lined with renal epithelium, and are imbedded in loose fibrillated tissue rich in cells. Occasionally, also, there is an admixture of striped muscle fibres in small quantities, but their occurrence is rare. In a considerable number of cases these tumours are separable from the surrounding renal tissue, and may be in part encapsuled. On section they are found to be mainly solid, but some of the cysts are often large enough to be seen with the naked eye. They are very malignant, and operations are but rarely successful in ultimately saving life.

All malignant growths of the kidney may form swellings of sufficient size to be felt by an examination of the abdominal wall, though in some instances the size of the swelling is rather dependent upon secondary hydronephrosis than on the actual bulk of the new growth. Hæmaturia is the most prominent symptom of all malignant tumours.

The most common **innocent** growth of the kidney is a cyst. Simple single cysts are occasionally met with in glands which are otherwise healthy; they rarely attain a considerable size, but sometimes contain as much as half a pint of fluid. The fluid is usually quite clear and watery, and often contains no urea. The cysts are apparently formed in connection with the renal tubules. They do not usually give rise to any symptoms.

Congenital cystic degeneration of the kidneys is occasionally seen in still-born children, and yet more rarely in those who survive their birth for a few years. Virchow has found in such organs an atresia or closure of the straight ducts which terminate in the papillæ, and suggests

that the occlusion is of inflammatory origin. In many cases, however, there is some associated malformation of the pelvis of the kidney or of the bladder or ureter, and it does not seem probable that the disease in question is inflammatory in its nature; it would rather appear to result from some irregularity in the development of the organ. The ureter is sometimes occluded at its junction with the renal pelvis. Such kidneys form large swellings, which push up the diaphragm and prevent expansion of the lungs. Their pelves and calices are large, and their surfaces are thickly studded with cysts of various sizes.

Multilocular cystic disease and general cystic degeneration are names used to indicate a form of renal disease in which each kidney is enormously enlarged and converted into a series of cysts, which are in no way the result of obstruction to the outflow of urine. In these cases there is no dilatation of the pelvis or calices, but the whole gland is converted into cysts, some as large as a pea or a nut, and others of much greater size.

The disease appears to be most common between the ages of thirty and forty, and is associated with a low specific gravity of the urine and the passage of a little albumen. Both kidneys are almost always affected, though one may be more diseased than the other. They may form tumours of great size, and may weigh several pounds.

Amongst the rarer tumours of the kidney may also be mentioned hydatid cysts and fibrous or fatty growths.

CHAPTER XLVI.

DISEASES OF THE BLADDER.

CYSTITIS.

Cystitis, or inflammation of the bladder, is chiefly met with as a complication of other diseased conditions of the genito-urinary tract, but may also originate from exposure to cold and wet, or during the course of one of the exanthemata.

In many cases it results from an extension of gonnorhœal inflammation from the urethra or prostate; in others, from retention of urine in patients with enlarged prostate or stricture of the urethra; whilst the irritation caused by calculi, the passage of instruments, the presence of new growths, and an irritating condition of the urine account for most of the remaining cases.

In its more **acute** forms cystitis is characterised by the appearances common to the inflammation of any mucous surface, namely, redness and swelling. These changes are accompanied by catarrh, and the consequent admixture of the urine with serum, mucus, and epithelial débris. In bad cases the inflammation may progress—ulceration may ensue, and a purulent catarrh be established. Occasionally, portions of the mucous lining slough, and sometimes false membranes of a "diphtheritic" character are formed.

Chronic cystitis is characterised by the formation of much stringy and viscid mucus, and by alkalinity of the urine. This state of the urine results from the action of a ferment present in the mucoid secretion, which causes

decomposition of the urea, and the formation of carbonate of ammonia. The alkaline urine in its turn keeps up the cystitis, and thus the two conditions act and react on each other. All alkaline urine contains micrococci and bacteria. It is a well-recognised fact that retention of urine is a very common cause of decomposition of that fluid, and in the opinion of some surgeons the decomposition always results from the passage of instruments and the conveyance of "septic" matter to the interior of the bladder. That this is a fertile source of cystitis and alkaline urine cannot be doubted, but it is certainly not correct to say that it is the invariable or even the common cause. Numerous cases of alkaline urine come under notice in which no instrument has been passed, and, to explain these, it has been suggested that mucus sticks about the urethra, and that the growth of organisms and decomposition extend along it from the meatus to the bladder. This, again, may be true of some cases, but there is no necessity to raise any such difficulties or supply such doubtful explanations. It is quite certain that micrococci can, and do, obtain access to the interior of the body independently of any wound or direct means of communication, and it is highly probable that they are incapable of doing much harm except when in contact with unhealthy tissues. There is no more difficulty in explaining the presence of micro-organisms in the inflamed bladder than in the pus of an acute abscess.

An examination of the bladder of a patient who has died with chronic cystitis generally reveals a contracted viscus with thickened walls, but it is evident that the appearances due to the chronic inflammation will often be masked by pathological changes resulting from the trouble which has itself been the cause of the cystitis—*e.g.*, stricture, enlarged prostate, &c.

The mucous membrane is swollen and thickened, with prominent veins and areas of congestion and submucous extravasation. Its colour is dark grey, with patches of purple or dusky red. Phosphatic deposits are often found

in places, and occasionally ulcers may be seen about the trigone.

The urine in such a case is turbid and alkaline, giving a foul ammoniacal smell. It is mixed with mucus, pus, and blood, and, on standing, deposits crystals of triple phosphate.

ENDEMIC HÆMATURIA.

The endemic hæmaturia of South Africa and of Egypt is now known to be due to the presence of a parasite, the

FIG. 115.

Bladder from a case of Bilharzia, showing the thickened and papillomatous mucous coat.

Bilharzia hæmatobia. The Bilharzia is a species of fluke in which the sexes are distinct. The male worm is flattened, and is 12–14 mm. in length; its posterior part forms a tube or gynæcophoric canal, into which the female passes during coition. The female is 16–19 mm. in length, and is cylindrical.

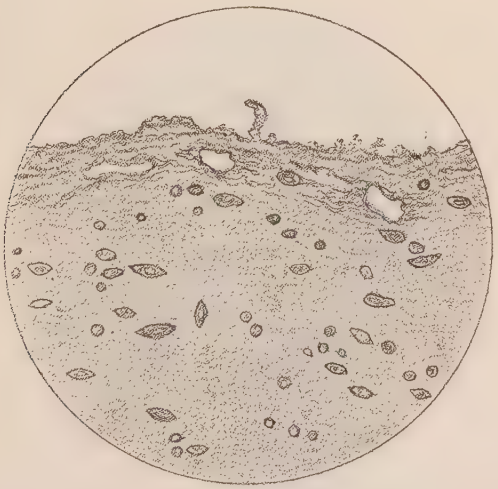
The worms are supposed to obtain entrance to the human body through the medium of drinking-water, and are found either in the portal vein or in the splenic, mesenteric, rectal, or vesical veins. As many as two or three dozen

females may be found in a single patient, but the male worm is very rarely seen.

The worms breed freely in the veins, and their ova penetrate into the neighbouring tissues, and especially into the urinary organs. The ova are oblong in shape, and 0.12 mm. in length; they are characterised by the presence of a projecting spine, which may be either terminal or lateral.

These ova, penetrating into the sub-epithelial tissues of

FIG. 116.



Section of a Bladder wall showing Bilharzia ova.

the kidney, ureter, or bladder, set up inflammation of the mucous membrane, and cause destruction of the epithelium, with resulting hæmorrhage and ulceration. In this way the mucous lining of the bladder may be extensively destroyed, and the vesical wall may become roughened by the growth of granulation tissue, which tends to assume a papillary form, and causes a general rough or villous appearance of the viscus. In the rectum also similar processes may occur, and large papillary growths, varying in

size from that of a pea to that of a walnut, may extend into the cavity of the bowel, and cause severe hæmorrhage and tenesmus. Such papillomatous growths I have found filled with ova of the parasite. I have also found the ova in the spleen and in the lung in the case of an Arab boy, whose viscera were kindly sent me by Dr. Mackie of Alexandria.

The ova do not further develop in the human body, but after being ejected from the bladder or rectum pass into another host (probably a mollusc) and in it develop into

FIG. 117.

A Bladder with a Sessile Villous Tumour.

cercaria, which in turn may grow into adult worms if ingested by man. The life of the parasite must be of considerable length, for ova may be passed and attacks of hæmaturia may recur for several years after the patient has left the country where the parasite is found.

TUMOURS OF THE BLADDER.

Tumours of the bladder may be divided into **innocent** and **malignant**. The latter are considerably the more common.

The innocent growths may be subdivided into papillo-

mata or villous tumours, mucous polypi, and fibrous polypi. Of these, the **papillomata** are the most common. The surface of one of these growths is covered with delicate branched processes, or papillæ, which float out in water, giving it a shaggy or "villous" appearance. Each papilla, on microscopic examination, is found to consist of a delicate central stalk of very fine, loose connective tissue, in which are embedded many round and oval cells. This stalk, which contains a central looped blood-vessel, is

FIG. 118.

Multiple Mucous Polypi in the bladder of a child.

covered by a layer or layers of oval or fusiform epithelial cells. The whole growth is usually sessile, being attached to the bladder by a broad base, but is sometimes distinctly pedunculated. On account of their extremely delicate structure, such tumours are liable to give rise to considerable hæmorrhage.

Mucous polypi are rare, and are met with only in children. They are composed of myxomatous tissue, such

as is found in the common mucous polypi of the nose. In some cases they are very numerous, covering the greater part of the vesical mucous membrane.

Fibrous polypi are also rare. They occur in children, and are liable to prove fatal by interfering with the passage of urine, and thus inducing disease of the kidneys.

FIG. 119.

A Bladder with a Large Cancerous Growth, the surface of which has ulcerated and is ragged and shreddy.

Malignant tumours of the bladder are more common than the innocent growths already mentioned.

Of the **carcinomata**, scirrhous cancer may be said never to occur in the bladder, whilst encephaloid growths are also rare. Epithelioma is often met with, and may originate on any part of the surface of the viscus. It chiefly attacks men past middle age, and is sometimes seen growing on two or more separate portions of the mucous membrane. The surface of an epithelioma usually ulcerates before the growth attains any considerable size, and the tumour itself, being macerated in the urine, becomes shreddy and roughened, forming what has been called "villous cancer." Such growths present, in their early stage, a very close

resemblance to the innocent villous tumours described above, but, unlike the latter, they tend to infiltrate the surrounding tissues, and, in rare cases, cause glandular affection and secondary tumours in the viscera. Their growth is slower than is usual in epitheliomata of many other parts, and often extends over a year or two.

Sarcomata are of less common occurrence in the bladder than carcinomata. They appear to behave as do sarcomata in other situations—infiltrating, breaking down, and occasionally disseminating. They differ in appearance

FIG. 120.

The Bladder of a Child, showing sarcomatous growths which caused death by obstructing the outflow of urine.

from the carcinomata in their greater bulk and more fleshy structure on section. Their surface also is not so ragged and shreddy as is that of an epithelioma.

The above short description of bladder tumours would be incomplete without some reference to the effects which they produce on the urinary organs. These effects differ much according to the nature of the growth.

The innocent tumours, as a rule, cause but little vesical irritation, and often no cystitis at all. The papillomata and mucous polypi chiefly give rise to trouble by the

hæmorrhage which takes place from their surfaces. This is often the only symptom which the surgeon finds to guide him, for, as a rule, they are so soft as to escape detection, both by the sound and by rectal examination. Any innocent growth, if it obstructs the orifices of the ureters or urethra, may give rise to further trouble, and produce secondary disease of the kidneys, with distension of their pelves and calices, and absorption of the renal tissue.

Malignant growths, on the other hand, usually cause much vesical irritation, and set up severe and intractable cystitis, with alkaline and foul urine mixed with blood and pus. In addition to this, such tumours are usually readily felt by instruments in the bladder, or by the finger in the rectum, for the wall of the latter viscus is often infiltrated. When a patient dies of a malignant growth in the bladder, suppurative nephritis is usually found at a post-mortem examination, and is readily explained by the extension of inflammation from the bladder, as well as by the occasional obstruction to the passage of urine.

CHAPTER XLVII.

DISEASES OF THE PROSTATE.

PROSTATITIS.

Acute inflammation of the prostate, or "prostatitis," is commonly the result of gonorrhœa, and arises from a direct extension of the inflammation from the prostatic urethra. It may also be set up by the passage of instruments.

Prostatitis causes much swelling of the whole gland, and is accompanied by great pain, frequency of micturition, and fever. In some cases the swollen gland interferes with the passage of urine and causes retention. In many patients the inflammation terminates in suppuration, with much brawny induration of the perineum. The pus, if left alone, may make its way in various directions. In favourable cases it bursts into the prostatic urethra, and is then evacuated with but little trouble. In other instances it passes backwards into the rectum. Sometimes, but fortunately rarely, it escapes into the cellular tissue around the prostate and there becomes diffused, setting up general cellulitis and suppuration. It is evident that rupture in the latter situation is fraught with more trouble and danger to the patient than when the pus escapes into the urethra or rectum.

Chronic prostatitis results from an acute attack, or from gonorrhœa, stricture, or exposure to cold and wet. It is accompanied by slight enlargement and tenderness of the gland, with gleety discharge, and is usually very chronic and troublesome to treat.

HYPERTROPHY OF THE PROSTATE.

Hypertrophy of the prostate is essentially a disease of late life, seldom or never commencing before the age of fifty years, and being much more common over sixty. It does not appear to have any definite cause.

The gland may be enlarged as a whole, all its three lobes being equally hypertrophied, or, whilst there is general enlargement, one or other lobe may be more hypertrophied than the remainder. Rarely, the increase in size affects one lobe alone.

A normal prostate is composed of fibrous tissue, gland tissue, and involuntary muscle fibre, but in the hypertrophied organ the normal proportions of the several constituents are seldom preserved. In most cases the fibrous tissue is increased far more than are the glandular and muscular structures, and the organ, consequently, is more dense and firm than natural.

The size of enlarged prostates varies greatly, but the slighter hypertrophies not infrequently cause quite as much trouble as do the more marked enlargements, and this is more particularly the case when the enlarged prostate encroaches upon the bladder cavity, and by its presence maintains a permanent condition of irritation in that viscus.

In many specimens of hypertrophied prostates definite **tumours** are found on section. These are usually firm, white, and fibrous, sometimes no larger than peas; in other cases as large as a walnut. They are surrounded by a capsule, and, on microscopic examination, are seen to be composed of fibrous and muscular tissue. Other tumours, not so white and dense, and not so definitely encapsuled, consist of a fibrous stroma in which is embedded gland tissue, whose acini are often distended so as to form minute cysts.

The effects of prostatic hypertrophy are seen in the urethra, bladder, ureters, and kidneys.

The urethra is in all cases lengthened in proportion to the size of the growth, and in some cases the prostatic portion of the tube measures two or three inches, instead of an inch and a quarter. On account of this, prostatic catheters are made longer than others. In addition to its increase in length, the urethra becomes more or less tortuous. Thus, if one lateral lobe be more enlarged than the other, it will cause the urethra to bulge towards the opposite side ; if the middle lobe be enlarged, the urethral floor will be pushed up from below so as to cause a con-

FIG. 121.

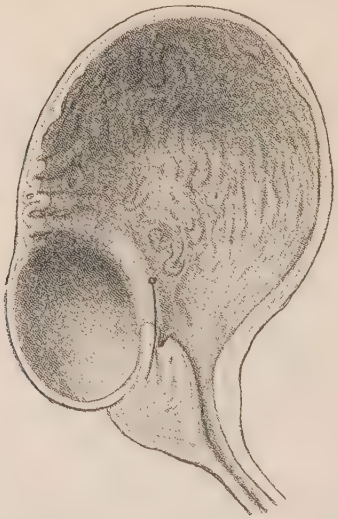
An Enlarged Prostate with an Hypertrophied Bladder. A sacculus has developed at the uppermost part of the bladder.

vexity in this situation. If there is much general enlargement, the urethra becomes compressed so that the lumen of its tube is narrowed and the passage of urine is interfered with. In this way retention may be caused.

In the **bladder** the urethral orifice is pushed upwards, so that it no longer occupies the lowest position in the erect posture, and thus predisposes the urine to collect in a pouch behind it. In some cases the orifice of the urethra is

surrounded by a ring of prostatic growth, which is most prominent when viewed from the bladder. On account of the obstruction offered to the passage of urine, the vesical walls hypertrophy, and the organ may subsequently become fasciculated and sacculated, as described in a previous chapter. When the middle lobe is much enlarged, it tends to become pedunculated and to extend towards the vesical cavity. It thus becomes a very prominent cause of difficult

FIG. 122.



Section of a Dilated Bladder, showing the pouching of the bladder-wall which occurs behind an enlarged prostate. A bristle has been introduced into the ureter.

micturition and retention of urine, for it not only lies directly over the urethral orifice, but tends to be thrust down as a valve over that orifice by the pressure of the urine in attempts at micturition.

As time goes on, and micturition becomes increasingly difficult, more and more urine is habitually retained, and this for several reasons. The more nearly the bladder is

emptied, the less efficaciously is it able to contract with force on its contents; and the more the urethral orifice is pushed up by the enlarged gland, the more does urine collect in the lowest part of the bladder behind the prostate. Again, when the enlarged median lobe acts as a valve, occluding the urethral orifice on any attempt at micturition, urination becomes possible only when, by the distension of the bladder and the traction on its walls, the median lobe is drawn upwards and backwards and removed from the urethral aperture. Even then, as the bladder contracts and empties its contents, the middle lobe again settles down and prevents the complete expulsion of the urine. But as the obstruction to micturition becomes greater, the bladder becomes habitually more and more distended, until finally, being always as full as it will hold, it ceases to act as a reservoir. The urine which flows into it from the ureters then constantly dribbles out through the urethra, and permanent incontinence therefore results. In time, a bladder which is thus chronically over-distended loses tone, and becomes paralysed or atonied.

The retention in its turn sets up cystitis, with alkalinity of the urine and the secretion of much sticky, viscid mucus. The urine is often very foul indeed, and in bad cases contains pus and blood derived from the ulcerated bladder-walls. Occasionally, phosphates are deposited in the alkaline urine, and form concretions on the mucous membrane. In other cases definite calculi may be formed.

The effect of the retention of urine and of the hypertrophy of the bladder-walls is **distension of the ureters and of the renal pelves and calices**. Pyelitis and nephritis may also be caused by extension of inflammation from the bladder.

In addition to the chronic retention of some of the urine in cases of enlarged prostate, acute and complete retention is liable to result from anything which causes congestion of the prostate and consequent swelling of the gland. Thus, a man who has always been able to pass his water, although

with difficulty, may have an attack of acute retention as the result of exposure to cold and wet, or of indulgence in alcoholic liquors. Either of these may cause prostatic congestion and swelling—conditions which are usually temporary and amenable to properly directed treatment.

TUMOURS OF THE PROSTATE.

Innocent tumours of the prostate, which consist of fibrous or glandular tissue and complicate general hypertrophy, have already been described; they are almost the only new growths of an innocent nature that are met with in this gland.

Malignant tumours are usually carcinomata, though sarcomatous growths have been observed. The carcinomata are of the glandular or spheroidal-celled variety, and are more often soft or "medullary" than hard or "scirrhous." They grow rapidly, and tend to cause the same complications as similar tumours arising in the bladder. Not infrequently they cause pressure on, or fungate into, the rectum.

PROSTATIC CALCULI.

The calculi which are of common occurrence in the prostates of old men have nothing to do with urinary calculi. They are developed in the glandular tissue, and form small concretions about the size of grains of barley, and seldom larger. They are commonly multiple, and by their mutual pressure become facetted. In most cases they give rise to no trouble, but in some cause difficulty of micturition, and more rarely suppuration in the prostate. They are composed almost entirely of phosphate of lime, with about 15 per cent. of animal matter.

CHAPTER XLVIII.

TUBERCULAR DISEASE OF THE GENITO-URINARY TRACT.

Tuberculosis may affect any part of the genito-urinary tract, and, as it is specially liable to attack many parts in the same patient, it is advisable to treat of the disease as a whole. It may commence in the bladder, kidneys, or testes, and is especially common in young adults.

The first morbid change in the **bladder** is the growth of small grey tubercles, such as are commonly seen wherever tuberculosis occurs. These, which are most numerous about the trigone, after a time break down and form circular superficial ulcers without any induration or thickening. By an extension of this process, the mucous lining is destroyed and general cystitis is set up.

In the **ureters** similar changes ensue, and these tubes become ulcerated and roughened; their walls also are usually much thickened. In some cases great obstruction is offered to the passage of urine, and occasionally the ureter is practically obliterated.

In the **kidneys**, the pelvis and calices are chiefly affected. Their mucous lining becomes swollen and thickened, and in time ulcerated, forming a soft pulpy membrane like a piece of sodden wash-leather. The discharge from the inflamed surfaces is at first mucoid, but very soon becomes purulent and blood-stained. The course of the disease differs much in different cases. In some, the urine finds difficulty in obtaining an exit on account of the swelling of the soft

tissues and obstruction in the ureter caused by the passage of the thick pus and mucus; the pelvis and calices consequently become distended, and the whole kidney greatly enlarged. Suppuration commonly extends to the kidney substance, and in time to the peri-nephritic tissue, causing

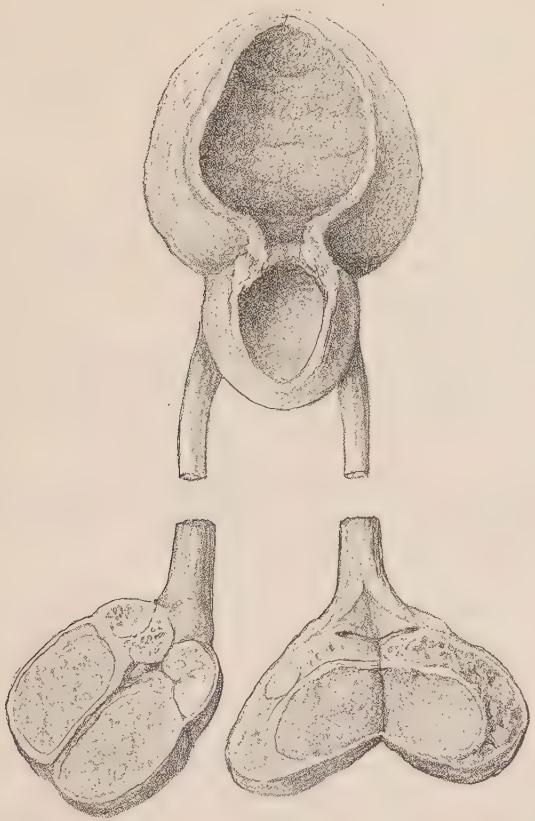
FIG. 123.

A Tubercular Kidney and Ureter. The kidney shows numerous cavities which were filled with caseous pus. The mucous lining of the ureter is roughened and ulcerated, and the whole tube is much enlarged.

in the latter situation the formation of a peri-nephritic abscess. This, in rare cases, discharges into the colon, but more commonly comes to the surface and bursts externally, the patient subsequently dying from exhaustion or from

destruction of the renal secreting substance. In other cases the inflammatory process gradually subsides, the

FIG. 124.



Tubercular Disease of the Testes, Vasa Deferentia, and Prostate. The testes are studded with numerous caseous nodules, the vasa deferentia are thickened, and the prostate contains a large abscess-cavity.

exudation diminishes, and many of the products of inflammation undergo caseation, forming a putty-like pultaceous mass. The kidney in such a case loses all functional

activity, and is practically placed outside the organism, just as much as is a caseous lymphatic gland. If the other kidney is healthy, and is capable of becoming hypertrophied to a sufficient extent, life may be prolonged indefinitely.

In many patients the disease commences as a deposit of tubercle in the renal cortex. From this, as from a centre, the mischief spreads, other deposits occur in different portions of the gland, and thus several caseous masses are formed. Any of these may burst into the pelvis, and from this the tubercle may extend to the ureter.

In the **prostate**, tubercle causes a general enlargement of the gland, with the formation in it of caseous masses and abscesses. These commonly come to the surface at the prostatic urethra or the neck of the bladder, and discharge by an irregular ragged aperture.

When the prostate is attacked, the **vesiculæ seminales** are also usually affected. They become enlarged, hardened, and nodular, and, on section, are found to contain caseous matter.

Tubercular disease of the **testis**, which is almost always present when the vesiculæ and prostate are affected, is described in a future chapter.

CHAPTER XLIX.

URINARY CALCULI.

The salts which are normally held in solution in the urine may be deposited from that fluid in the form of concretions or calculi, and may then be retained in any part of the urinary tract.

The most common calculi are those composed of uric acid, of urates, of oxalate of lime, and of fusible phosphates.

Calculi composed of **uric acid** or **urates** are met with chiefly in the children of the poor, and in men of the gouty or uric-acid diathesis. In children they appear to result from feeding with indigestible or improper food, which results in the production of undue quantities of waste matter and excess of work for the excreting organs. The urine consequently becomes highly acid and concentrated, and the solid matter being more than can be properly held in solution, is deposited as a sediment in the renal tracts.

The ingestion of excessive quantities of food or of alcoholic liquors, combined often with deficient exercise, a torpid liver, an unhealthy skin, and constipated bowels, brings about the same result. More work is thrown on the kidneys than should fall to their share, the urine becomes concentrated, and calculi are formed. It appears probable that mere concentration of urine is not, however, enough in itself to produce calculi, and that the highly acid urine first causes a catarrh of the renal tubes. The crystalline deposit is supposed to be formed in the renal epithelial cells, and to extend from them to the lumen of the tubes.

Oxaluria, or the presence of **oxalate of lime** in the urine in excessive quantities, can hardly be regarded as having been at present satisfactorily explained. It has been described for many years as occurring in connection with a definite diathesis, the so-called "oxalic-acid diathesis," in patients who are in a broken-down or debilitated state of health, who are over-worked or worried, with much nervous depression, &c., but at the present time there are many who think that there is no definite diathesis associated with the formation of oxalate of lime, and consider that its presence in the urine is due to the ingestion or failure of assimilation of vegetables and fruits. Other authors are of opinion that oxalic acid is formed by the decomposition of uric acid, and consider that its production is only a variation or modification of the uric-acid diathesis. Whatever may be the final conclusions, it is certain that oxaluria and oxalate-of-lime calculi are by no means limited to debilitated or broken-down patients, and that oxaluria often alternates with the presence of uric acid and urates in the urine.

RENAL CALCULUS.

Most of the calculi which are formed in the kidneys escape from the gland while yet small, and are subsequently voided in the form of gravel. Some, however, remain behind. The effects produced by a renal calculus depend much upon its position and mobility, and to a less extent on its size. If formed and retained in the cortex, it may become encysted and fixed, shut off from the urinary tract, and so give rise to little or no trouble. If, on the other hand, it passes into the pelvis, it is liable, by its constant movement, to cause **pyelitis**, with, in time, hæmaturia and pyuria, whilst, by occasionally blocking the orifice of the ureter, it may bring on attacks of renal colic and cause distension of the pelvis and calices. The more freely movable it is the more pain it will cause.

The worst, however, that can result from the presence

of a calculus in the kidney is the blocking of the ureter by the impaction of the stone. The urine is then retained in the renal pelvis, and **hydro-nephrosis** results, the calices becoming distended and the glandular tissue being absorbed by pressure.

In many cases the irritation caused by the stone results

FIG. 125.

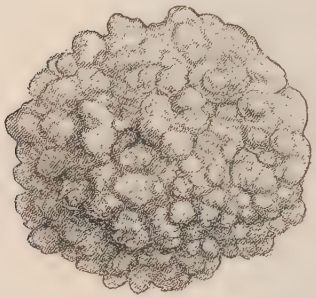
A suppurating Kidney with a Calculus impacted at the Orifice of the Ureter. The Calices are dilated, and the renal tissue atrophied. The kidney was very large, and full of pus.

in suppuration of the pelvis and calices, and pyuria. The pus usually has some difficulty in escaping, on account of inflammatory swelling of the mucous membrane of the pelvis and ureter, and the kidney consequently is distended by it, and becomes in time a huge multilocular abscess-cavity, often producing a considerable swelling in the lumbar region. When the renal pelvis is distended by pus, the condition is named **pyo-nephrosis.** In cases such as these the suppuration may extend to the neighbouring cellular tissue, and cause the formation of a **peri-nephritic**

combination. Such stones are smooth, round, or oval, fawn-coloured or earthy on section, not laminated, and not so hard as the uric-acid calculi.

Oxalate-of-lime stones are seldom more than two ounces in weight, and most of them are much below this. In shape they are usually irregularly rounded, with numerous nodular projecting portions, which give them a rough and tuberculated appearance. On section, they are found to be very hard and tough, and of brownish or green

FIG. 128.



An Oxalate-of-Lime Calculus.

FIG. 129.

Section of an Oxalate-of-Lime Calculus, showing the irregular wavy laminæ.

colour. They are generally laminated, but the concentric laminæ, instead of being regular and ring-like, as in uric-acid stones, are usually wavy and jagged. Oxalate-of-lime calculi are soluble in hydrochloric acid.

Fusible calculus, or calculus composed of phosphate of lime with phosphate of ammonia and magnesia, is the commonest form of phosphatic stone, and sometimes attains a great size. It is of a dead-white colour, soft, light, smooth, and not laminated on section.

Cystine calculi are oval, finely crystalline on the surface, and, when recently extracted, of a yellow, honey colour. They are soft and friable, not laminated on section, and are readily soluble in ammonia. These calculi are

remarkable for the change in colour they undergo after exposure to the light for some months, for it will be found that they then gradually assume a delicate emerald-green hue.

Other forms of calculi are rare, and do not require separate description. They are composed of xanthic oxide, phosphate of lime, carbonate of lime, and ammonio-magnesium or triple phosphate.

Many calculi are not composed throughout of the same substance, but are formed of concentric layers of different formation. Thus, it is common to find a uric-acid nucleus surrounded by a ring of phosphates, and this in its turn coated with another layer of uric acid or urates, the whole perhaps surmounted by another phosphatic coat. In other cases oxalate of lime may alternate with uric acid, urates, or phosphates.

The alternation of the phosphatic and uric-acid layers is explained by the differing reactions of the urine. When the latter is acid, the calculus increases in size by the deposit of uric acid; but when cystitis has caused the urine to become alkaline, then the phosphates which are normally held in solution become deposited.

Effects of a stone in the bladder.—A calculus in the bladder always sets up a certain amount of cystitis. To a limited extent the latter is proportionate to the roughness and size of the stone, but it is nevertheless true that in many instances small and perfectly smooth stones cause infinite pain and vesical irritation, whilst in other patients large and rough stones elicit but few symptoms. This is to be attributed to the different idiosyncracies of the patients themselves; children usually suffer more than adults. The changes that occur in cystitis have already been described; but it must be pointed out that the obstruction to micturition caused by the thick and viscid mucus is probably one of the causes of the hypertrophy of the bladder which is a common result of a calculus. This hypertrophy is also probably induced by the occasional

obstruction of the urethra by the stone—a circumstance which is most likely to happen at the end of the act of micturition.

In cases of long standing, sacculi may be developed in the hypertrophied viscus. These are of much importance, for in them stones may lodge, or, if lithotrity be performed in the sacculated bladder, great trouble and danger may arise from fragments becoming encysted. The changes that ensue in the ureters and kidneys are such as have already been described under the head of "Surgical Kidney.

CALCULI IN THE URETHRA.

Small calculi are often passed by gouty people who suffer from gravel in the urine, and the large majority of such stones are not retained in any part of the urethra.

The impaction of a calculus is of much more frequent occurrence in children than in adults. The usual place for a stone to lodge is the fossa navicularis, for the very good reason that the meatus is the narrowest point in the whole urethra. Such impaction, indeed, is the most frequent cause of retention of urine in children, and in any case of the kind ought to be at once suspected.

The local effect of the lodgment of a stone in this position is a general œdema of the prepuce, penis, and often of the scrotum, and sometimes urinary extravasation, which probably results from ulceration of the mucous membrane. Although retention is marked, the urethra is often not completely plugged, and the urine consequently may dribble away.

In adults a calculus may lodge behind a stricture. In such cases the urethra usually becomes ulcerated, a perineal abscess forms, bursts externally, and establishes a perineal fistula. In rare and fortunate cases the stone escapes with the pus.

CHAPTER L.

DISEASES OF THE TESTIS.

MALPOSITION OF THE TESTIS.

The testis may be arrested in any part of its passage from the lumbar region to the scrotum. First, it may never leave this region, but may remain permanently in close proximity to the kidney. Secondly, it may pass as far as the internal abdominal ring, but fail to enter the inguinal canal. Thirdly, it may pass into the inguinal canal, but fail to traverse the external ring. Fourthly, it may pass through the ring, but not descend into the scrotum. The causes of this retention are variously stated. It is probable that the most frequent cause is intra-uterine peritonitis and the formation of adhesions between the folds of peritoneum in the neighbourhood of the gland, but an unusually small external ring, and a want of power in the gubernaculum testis, are believed to act as causes of an incomplete descent. Shortness of the vas deferens and an unusually large epididymis have also been claimed as causes of retention. The term "cryptorchid" is applied to men in whom both testes are retained, and that of "monorchid" to those in whom only one gland is undescended.

There has been much controversy as to the condition and development of a retained testis, and it is now tolerably clear that, whereas in the great majority of cases the testes are undeveloped and the patient sterile, in a very few the

gland is active, and semen is normally secreted. It is generally considered that the testis is less likely to be undeveloped if retained in the abdomen than if subjected to the pressure of the muscles by retention in the inguinal canal, but this is very doubtful. With regard to virile power, it may be said that it is almost always present, even when both testes are retained and the patient is sterile. In all cases of undescended testis the corresponding half of the scrotum as well as the tunica vaginalis is imperfectly developed. According to the researches of Dr. Joseph Griffiths, the retained testes are characterised by the disproportionate amount of connective tissue they contain, and by the thickening of the walls of the tubules and the small development of epithelium.

Occasionally, the testis, instead of being retained as above described, descends into unnatural positions, and may thus pass into the perineum or, perhaps, through the crural ring into the femoral canal. I have also seen a case in which, after its transit through the external ring, the gland passed outward into Scarpa's triangle.

Complications of retained or misplaced testis.—A testis which is retained in the inguinal canal is constantly liable to attacks of orchitis caused by the contraction of the abdominal muscles during any violent exertion, and it is by no means uncommon for the gland in such cases to be forced through the external abdominal ring. Such extrusion is merely temporary, and the testis almost at once returns to its previous position. In the perineum also, the gland is greatly exposed to injury, and thus is rendered liable to frequent attacks of orchitis. It must further be remembered that when the testis is retained it is in close proximity to the peritoneum, and that, consequently, inflammation commencing in the testis may extend and cause peritonitis. The gland when retained is liable to be the seat of orchitis in cases of gonorrhœa or of mumps, exactly as it is when in its normal position.

Retention of the testis is generally believed to predispose

it to malignant disease in a marked degree, and the explanation that is commonly given is that imperfectly developed organs are more liable than healthy ones to be thus affected, and that the irritation and frequent attacks of orchitis induced by pressure in the inguinal canal are likely to act as exciting causes. Considering the comparative frequency of undescended testis, I am myself by no means convinced that it is attacked by malignant growth in a larger percentage of cases when in the inguinal canal than when in the scrotum.

Interstitial inguinal hernia is a common complication of undescended testis, and generally results from failure in the normal process of closure of the funicular portion of the tunica vaginalis. Mr. Curling and other writers on the subject believe that hernia is much more likely to complicate a late descent of the gland than is its simple retention, for in some patients testes which are retained for months after birth subsequently descend. In such cases the intestine may be found adherent to the testis. Occasionally, hydrocele complicates retention of the testis.

Torsion or axial rotation of the testis.—Attention has only very recently been directed to this condition by the description of a case by Mr. G. Nash in 1891, and by the subsequent record of other cases by various surgeons, chiefly in England. As the name implies, the morbid condition is a twist of the cord and epididymis round their long axes so that the vessels of the cord are obstructed, and the testis first becomes congested, and, if unrelieved, gangrenous. The accident appears to occur chiefly in boys and young men, and almost invariably affects a testis which is undescended or whose descent is complicated by a congenital hernia. Either gland may be involved, and the twist may be either outwards or inwards, but it is quite uncertain how the twist is caused. Clinically, the history of these cases is very like that of a strangulated hernia, there being a sudden swelling accompanied by pain and sickness, and much tenderness of the affected part. It

appears that even if such a testis does not become gangrenous, it subsequently undergoes atrophy, so that removal of the twisted gland is generally advisable.

Eunuchs.—A eunuch is a man who has either been deprived of the testes in childhood or in whom they remain undeveloped although normally placed in the scrotum. In either case the growth of hair and the development of the larynx, &c., which normally characterise puberty, do not occur, and not only do the external genital organs remain like those of a child, but the prostate gland is similarly undeveloped.

INFLAMMATION OF THE TESTIS—ORCHITIS.

Acute orchitis (as apart from epididymitis) most often results from injury, but is also caused by exposure to cold or wet, and is met with in cases of mumps and in gout, as well as, more rarely, in rheumatic subjects and in patients suffering from acute febrile disorders.

The inflamed testis presents the appearances common to all acute inflammations, but, on account of the denseness of the tunica albuginea, is unable to swell to any extent, and thus becomes proportionately painful. The inflammation rarely terminates in suppuration, the inflammatory products being readily removed by the numerous lymphatics of the part. Abscess and even gangrene have, however, been known to occur. In some cases the orchitis causes so much damage to the glandular epithelium that the testis subsequently becomes completely atrophied.

Chronic orchitis may result from an acute attack which has not undergone complete resolution, but, as it is in simple cases always combined with some inflammation of the epididymis, it is not necessary to further describe it here.

EPIDIDYMITIS.

Epididymitis, or inflammation of the epididymis, is almost always the result of some irritation or inflammation of the urethra. The epididymis is also commonly

affected in orchitis, and amongst the causes of epididymitis may therefore be enumerated those which have already been mentioned in connection with the latter subject.

Inflammation of the urethra, in whatever way excited, is liable to cause epididymitis, and thus the latter may complicate the urethritis caused by gout, the passage of gravel, the use of catheters, &c. Gonorrhœa, however, being the most common cause of urethritis, is also the most common cause of inflammation of the epididymis, and it may further be said that the epididymitis which results from an acute gonorrhœal urethritis is itself likely to be acute, whilst that which arises from a chronic urethritis or gleet is more likely to be chronic.

The epididymitis which results from inflammation of the urethra has commonly been spoken of as "metastatic," a term which implies that the inflammatory process is transferred from the one part to the other; it has also been called "sympathetic." There is every reason to believe that the affection of the epididymis is the result of an extension of the inflammation from the prostatic urethra to the common ejaculatory ducts, and thence along the vasa deferentia. This opinion is borne out by the fact that epididymitis is especially liable to result from the inflammations of the deeper portions of the urethra rather than from those of the anterior parts of that tube.

Acute epididymitis is attended with much swelling, which commonly extends to the spermatic cord and the testis; the epididymis is also extremely tender and painful. In these cases, as well as in acute orchitis, the inflammation frequently extends to the tunica vaginalis, and results in the condition known as "acute hydrocele." This is nothing more than an inflammatory exudation into the cavity of the tunica vaginalis, and is accountable for most of the swelling which is more commonly attributed to the testis itself. In acute epididymitis the scrotum is liable to become red and swollen, and in some cases is greatly enlarged by œdema.

Chronic epididymitis and orchitis may result from an acute attack, or may originate in a chronic inflammation of the urethra. They are perhaps more often dependent upon stricture and its accompanying urethritis than upon any other cause.

In such cases the epididymis becomes enlarged, hard, and knotty, from the formation in it of fibrous tissue developed from the inflammatory exudation. The testis also is swollen and tender, the cord is enlarged, and the tunica vaginalis contains some serous fluid. In cases of long standing the contraction of the newly formed fibrous tissue causes obliteration of the seminal tubules or of the vas itself. Atrophy of the gland also may result, as in acute orchitis. In some cases the inflammation terminates in suppuration, an abscess forms, the skin becomes adherent, and the pus is discharged. Occasionally, after the abscess has burst, the testis tends to protrude through the aperture, and the condition known as "**hernia testis**" is produced. This protrusion is generally associated with an unhealthy condition of the gland itself, and is much more common in connection with tubercular disease of the testis than with simple chronic orchitis.

TUBERCULAR, STRUMOUS, OR SCROFULOUS DISEASE OF THE TESTIS.

These are the various names applied to a chronic form of orchitis which exhibits in all its phases the characteristics common to tubercular inflammations.

It has already been mentioned that this affection is frequently an accompaniment of tubercular disease of other portions of the genito-urinary tract, and, as might be expected, it is seen chiefly in unhealthy or strumous subjects, and is more common before than after middle age. There is a great tendency for both testes to be diseased, though one may show symptoms of the affection long before the other is implicated. Tubercular orchitis usually commences as a swelling of the epididymis, and at first

causes no pain. The epididymis becomes hard and knotty, and, as it increases in size, forms a semicircular or crescentic mass behind the testis (see Figs. 124 and 130).

The early extension of the inflammation to the tunica vaginalis is signalised by the formation of a hydrocele, which often renders an accurate examination of the diseased parts more difficult than would otherwise be the case.

FIG. 130.

A Tubercular Testis. The epididymis is specially enlarged, and forms a crescent-shaped swelling behind the testis. The tunica vaginalis was distended with fluid.

As the inflammatory process progresses, the swollen epididymis softens at one part, and the superjacent skin becomes red, adherent, and thinned, and finally bursts, giving exit to a few drachms of pus. In the course of the disease the testis also becomes enlarged and nodular, then soft, caseating, or suppurating ; fresh abscesses form, and in many cases, through the sinuses which result, a protrusion or hernia of the testicular substance ensues. The spermatic cord, as a whole, is thickened, but the vas deferens

is especially hard and swollen. The disease may extend to the vesiculæ seminales, the prostate, or other parts of the genito-urinary tract, but in the majority of patients such a serious extension is not seen, and whilst in many the inflammatory process subsides without ever having arrived at suppuration, in others, after the pus has been discharged, the sinuses slowly heal, and the disease comes to a natural termination.

If the parts themselves be examined at various stages of the process, it will be seen that strumous disease commences in an inflammatory exudation into the connective tissue of the epididymis, combined with a catarrh of the ducts. The inflammatory products evince a great tendency to undergo fatty degeneration, and thus the tubes and connective tissue alike become filled with a caseous pulp. As the process extends to the testis and cord as well as to the skin, those structures also become involved in a similar destruction, and in many instances a little ill-formed pus is produced.

A section of such a testis shows a general enlargement of the gland, with several areas of inflammatory condensation, of caseous matter, or of pus. Towards the epididymis these areas tend to coalesce and unite with the caseous matter, which is here more especially abundant. The tunica vaginalis contains a varying amount of clear straw-coloured fluid, the tunica albuginea is thickened in patches, the cellular tissue of the spermatic cord is infiltrated and œdematous, and the vas deferens is enlarged to some five or six times its natural size, whilst its lumen is filled with caseous matter.

When an abscess bursts and exposes the interior of a broken-down testis, there is a considerable tendency to extrusion of the latter, together with some of the unhealthy inflammatory or granulation tissue which surrounds and infiltrates it. The protrusion is liable to become increased by the retraction of the skin, and where the cutaneous opening is large, and the skin not closely adherent

to the deeper parts, the retraction may result in a complete uncovering of the testis. In such a case the whole gland may be extruded.

A microscopical examination shows that the process is essentially tubercular, for typical tubercle may be found in all the diseased parts. The epithelium at first proliferates and fills the lumen of the tubes, but is in course of time slowly destroyed by an extension of the tubercular process to the glandular tissue itself. It appears probable that the disease always commences in the intertubular connective tissue, and subsequently extends to the epithelium. Sections of the vas deferens show a similar growth of tubercle in its walls, and in advanced cases partial obliteration of its lumen by a growth of granulation tissue and tubercle. As in the lungs and other parts, the process may spontaneously cease, and the caseous matter either become encapsuled, or, in process of time, undergo calcareous changes.

Miliary tubercle of the testis may also be seen in the form of typical grey granulations in cases of general tubercular dissemination, but does not require special description.

SYPHILITIC DISEASE OF THE TESTIS.

The testis may become the seat of syphilitic inflammation at almost any time after the development of constitutional symptoms. The gland may be the seat of either (1) a diffuse interstitial inflammation, or of (2) one or more gummata.

Interstitial orchitis occurs earlier than the gummatous form, and is commonly seen as a late secondary lesion; it often attacks both testes, and is characterised by a painless swelling of the whole testis, which becomes firm, heavy, of an oval shape, flattened from side to side, smooth on the surface, and not tender. There is frequently loss of testicular sensation. The epididymis is not noticeably affected, and the spermatic cord, although swollen, is not definitely

thickened. The tunica vaginalis usually contains an excess of fluid.

On section of such a testis, the tunica albuginea is found to be much thickened, and the fibrous septa which extend from it into the gland appear more prominent and dense than is normal. The gland itself is tough and fibrous and the testicular substance diminished. The fluid in the tunica vaginalis is clear, and in some cases the opposed surfaces of the serous membrane are united by adhesions. A microscopical examination shows a growth in the intertubular connective tissue, at first of leucocytes and of new blood-vessels, afterwards of spindle cells, and finally of fibrous tissue.

The termination of diffuse orchitis differs in different cases. In some, especially those which are not subjected to treatment, the seminal tubes are compressed by the contraction of the dense fibrous growth, and their epithelium, after undergoing fatty changes, is gradually cast off. By a continuation of this process the whole of the gland may be in time destroyed, and the shrinking of the fibrous tissue finally reduces the testis to a small hard mass with no trace of secreting structure. When, however, the disease is treated, the young connective tissue is commonly quickly removed, and the testis again returns to its normal size and function. In some cases portions only of the gland are affected by this form of interstitial inflammation, and the changes above described are then limited to one or more lobules.

Gummatous orchitis.—When syphilitic inflammation attacks the testis in an old or broken-down subject, or when it makes its appearance as a late tertiary symptom, the inflammatory products are liable to caseate and to form gummatous masses. These tend to implicate the tunica albuginea and the tunica vaginalis, to extend to the skin, and finally to burst, giving exit to the blood-stained mixture of cheesy matter and pus characteristic of gummata in other parts, and leaving a circular cavity in

the gland. In some cases of gummatous disease of the testis, the opening in the skin becomes the site of a hernia of the testis, such as occurs in simple chronic and strumous orchitis.

Section of a gummatous testis usually shows that the gland is the seat of both the diffuse orchitis described above and of the gummatous form. The processes in each are at first alike, and consist of an exudation of leucocytes and a growth of young spindle-celled connective tissue. In the gummatous variety this young tissue breaks down and forms the caseous pulp already alluded to. Gummata in the testis are usually multiple, varying in size from a pea to a chestnut. They are at first firm, yellowish-white, and elastic, often presenting a definite outline and a capsule of fibrous tissue. In their later stages they are caseous or suppurating.

In addition to the above well-recognised forms of syphilitic orchitis, Mr. Jacobson (in his article in Holmes's "System of Surgery") alludes to a chronic inflammation of the epididymis in cases of acquired syphilis, and to gummata in patients who have inherited the disease. Both of these conditions are, however, certainly rare.

CHAPTER LI.

TUMOURS OF THE TESTIS.

Tumours of the testis may be either innocent or malignant, but the latter are, unfortunately, by far the most common.

Malignant growths are either sarcomatous or carcinomatous, the sarcomata being more often seen than the carcinomata.

SARCOMA.

Sarcoma of the testis, according to Mr. Butlin, is met with most frequently under the age of ten, or between thirty and forty. Its origin is frequently attributed to injury, and its growth is very rapid. The testis enlarges without pain, but to a great extent maintains its normal shape, and forms a highly elastic swelling, which gives a deceptive sense of fluctuation ; normal testicular sensation is often lost. The skin is stretched in proportion to the size of the growth, but hardly ever becomes itself implicated or even adherent. If it does become involved, the tumour subsequently fungates as a bleeding mass, to which the name of "fungus hæmatodes" was formerly given. In the later stages, the spermatic cord is enlarged, and masses of infiltrated glands may be felt in the iliac and lumbar regions. The general health is by this time seriously affected, the patient becomes much emaciated, and dies from implication of the viscera.

An examination of the testis in an early stage shows

that the growth commences most commonly in the posterior portion of the gland, and that the testicular structure itself is often spread over the anterior surface of the tumour. The tunica albuginea at first stretches, but after a time gives way, and may thus allow hæmorrhage to take place into the cavity of the tunica vaginalis. This is an occurrence of some clinical importance, and it is well to remember that hæmatocele may complicate a sarcomatous growth, for in cases in which the diagnosis is difficult the

FIG. 131.

Section of a Large Sarcomatous Testis with Hæmatocele of the Tunica Vaginalis.

discovery of blood in the tunica vaginalis might otherwise lead to error.

On section, a sarcomatous testicle is very soft and pulpy, either opaque-white, pinkish, mottled, or gelatinous and semi-transparent; the spermatic cord may be infiltrated with similar growth. The iliac and lumbar glands often form a series of immense tumours, and the liver, kidneys, lungs, and other parts may be infiltrated with numerous secondary masses.

A microscopical examination usually shows a large, round-celled growth; but in some cases the cells are oval or

spindle-shaped, in others mixed. The round-celled tumours grow the most rapidly, and occasionally involve both testes.

CYSTIC TUMOURS OF THE TESTIS.

New growths of the testis containing cysts are of common occurrence, and being closely allied to the sarcomata, are best considered in conjunction with them. These tumours appear to originate in the hilum of the testis, and, although

FIG. 132.

Section of a Cystic Sarcoma of the Testis.

alike as far as the formation of cysts is concerned, differ materially in the formation of their more solid parts. They have lately been investigated by Mr. Eve, and, as his conclusions appear to be more satisfactory than those commonly accepted, I shall venture to quote them. He says that there are three forms—(1) **cystic fibroma**, (2) **cystic myxoma**, (3) **cystic sarcoma.**

In the first class the stroma is composed of fibrous tissue, with some admixture of cartilage, and occasionally of unstriped muscle. The cysts are commonly lined with spheroidal or flattened epithelium, but sometimes with columnar cells, which may be ciliated.

The cystic myxomata are composed of mucous and fibrous tissue, with cysts usually lined by columnar epithelium.

In the sarcomatous growths, also, the cysts are usually lined by columnar epithelium, which here again may be ciliated. Mr. Eve considers that the cystic tumours are developed from the remains of the Wolffian body, and thinks that the cysts also are of new formation; other authors generally are inclined to consider the cysts as developed from a dilatation of the ducts of the rete testis.

FIG. 133.

Section of a Portion of a Cystic Fibroma of the Testis. One of the cysts, lined by columnar epithelium, lies to the left of a rounded nodule of cartilage. Below and to the left side of the cyst is a stroma of fibrous tissue. (Crouch, $\frac{1}{2}$ in.)

It is evident, from a consideration of the great structural difference displayed by cystic tumours of the testis, that similar difference must be expected in their rapidity of growth and clinical course. Thus, a growth whose stroma is chiefly sarcomatous will develop as a sarcoma in spite of the cysts it contains, whilst the fibrous and myxomatous tumours will not only grow more slowly, but will also run a less malignant course. An examination of the testis in

these cases shows a corresponding difference in appearance, the sarcomatous tumours having the characters already described, whilst the fibrous or myxomatous growths display the appearances common to such tissues. The cartilage, when present, is commonly scattered in nodules throughout the section, being sometimes abundant, at other times very scanty. The cysts are commonly small, their usual size being about that of a pea. They contain blood-stained fluid, which is usually viscid, but sometimes thin and serous.

CARCINOMA.

Carcinoma of the testis is almost invariably of the encephaloid variety. Instances of the scirrhous form are very rare indeed, but there is one good specimen of such a growth in the museum of St. Bartholomew's Hospital.

In the rapidity of its growth, its implication of lymphatic glands, and its tendency to affect the viscera, encephaloid cancer does not differ at all from the more malignant forms of sarcoma; the clinical course of the two is indistinguishable, but, whereas sarcoma is sometimes met with in young children, carcinoma occurs only in adult males, commonly over thirty-five years of age. A section of a carcinomatous testis presents the same opaque-white, soft, brain-like mass seen in the most rapidly growing sarcomata, but the mixture of cartilage and myxomatous tissue and the formation of cysts do not occur in the encephaloid cancers.

Microscopical examination shows the usual alveolar stroma with masses of epithelial cells common to all carcinomata.

INNOCENT TUMOURS.

Innocent tumours of the testis are rare. They comprise enchondromata, fibromata, and dermoid cysts.

Pure **enchondromata** are very uncommon, though, as already described, cartilage may occur in connection

with cystic tumours. The cartilage may have either a fibrous or myxomatous basis, but is sometimes purely hyaline. Such tumours grow slowly, and do not tend to affect the lymphatic glands or the viscera.

Fibromata of the testis are still more rare, there being but two or three cases on record.

Dermoid Cysts of the testis are more common than either the cartilaginous or fibrous growths. They are of congenital origin, but may subsequently increase in size. The cyst-wall has the structure of true skin, and the cavity contains sebaceous matter, hair, and epithelial scales. In some cases these cysts have been found to contain cartilage, teeth, and bone.

Psammoma has been described by Virchow, and I have seen one case where the growth was apparently developed in connection with the visceral layer of the tunica vaginalis. Such tumours are of extreme rarity.

CHAPTER LII.

HYDROCELE, HÆMATOCELE, AND VARICOCELE.

A HYDROCELE is a sac containing fluid in connection with the testis or its ducts, but of such sacs there are several varieties.

VAGINAL HYDROCELE.

A vaginal hydrocele, or hydrocele of the tunica vaginalis, is the most common form, and consists of a distension of the tunica vaginalis with fluid. These hydroceles are of very frequent occurrence, and may develop at any age. With respect to their cause there is but little to be said, for although hydrocele may complicate any of the various forms of chronic orchitis, there is in uncomplicated cases no evidence of any inflammatory origin, the effusion appearing to be simply passive.

The **fluid** of a vaginal hydrocele is of a pale yellow, amber, or straw colour. It is thin and watery, with a neutral reaction, and a specific gravity of from 1020 to 1025. It contains about 6 per cent. of albumen, with a considerable quantity of fibrinogen, but no fibrin ferment, and does not coagulate spontaneously. In cases of long standing the fluid may present a bright and sparkling appearance caused by the presence of cholesterine crystals. In addition to these, the microscope shows merely a few epithelial cells.

The **tunica vaginalis** is in most cases simply distended, and shows no structural change, but in patients who have

long been the subjects of hydrocele, and especially in those who have frequently been tapped, the serous membrane is liable to become opaque and greatly thickened by a deposit of fibrin on its inner surface and by growth of fibrous tissue in its walls. Sometimes adhesions are formed and partially subdivide the cavity, and occasionally calcareous matter is deposited in the lining membrane. In some cases small fibrous bodies are found loose in the sac, and appear to have originated in the fibrinous exudation which is liable to follow tapping or other injury, thus closely simulating the melon-seed bodies developed in bursæ and ganglia. The hydatid of Morgagni, again, is sometimes enlarged, and attached by a pedicle of considerable length which allows of its free movement. To the irritation produced by this structure the causation of hydrocele is sometimes attributed, but the hydatid is often found pedunculated in the absence of hydrocele, and is not commonly pedunculated when the latter exists. The shape of a vaginal hydrocele is that of a pear with the stalk uppermost; its size varies within the widest limits.

The **testis** is almost always at the posterior and lower part of the sac. It is sometimes a little flattened by pressure, but its structure is not necessarily thereby affected. The connective tissue of the scrotum and cord may be slightly thickened, and the cremaster muscle is frequently hypertrophied to compensate for the extra work it is called on to perform.

Congenital hydrocele and **infantile** hydrocele are simply varieties of the vaginal form in which the funicular portion of the serous membrane is either patent or only partially occluded.

In the congenital variety there is a direct communication between the tunica vaginalis and the peritoneal cavity, and fluid will pass from one to the other according to the position of the patient or the application of pressure to the scrotum. The communication is in some cases free; in others the fluid can only with difficulty be squeezed out of

the sac drop by drop. Hernia may complicate this condition, but is only exceptionally present.

Infantile hydrocele is the name given to a hydrocele in which the fluid is not confined to the tunica vaginalis proper, but extends a variable distance along an unobliterated funicular portion of the serous cavity. Hydrocele in young children is often of this form: it possesses no special importance.

Inguinal hydrocele is the name applied to a hydrocele of the tunica vaginalis occurring as a complication of an undescended testis, and forming a cystic swelling in the inguinal region.

ENCYSTED HYDROCELE.

An encysted hydrocele is one in which the fluid is contained in a sac separate from that of the tunica vaginalis testis. There are three chief varieties—(1) encysted hydrocele of the epididymis; (2) encysted hydrocele of the testis; (3) encysted hydrocele of the spermatic cord.

Encysted hydrocele of the epididymis is, of these, much the most common. It is usually found in the substance of the upper part of the epididymis, and is often placed between the latter and the testis, the vasa efferentia being spread out over the sac. The fluid is commonly quite colourless, watery, and limpid, containing alkaline carbonates, chloride of sodium, and a mere trace of albumen. Sometimes, on the other hand, the fluid is opalescent or milky, and, on microscopical examination, is found to contain spermatozoa, a circumstance which has given rise to the term "**spermatocele.**" The cyst-wall is always very thin and delicate, being composed of connective tissue lined with flattened endothelium. The sac is usually globular, and varies in size from that of a hazelnut to an orange, the latter size being quite exceptional. The testis lies below and in front. The presence of spermatozoa in these hydroceles has led to much controversy and speculation, and until recently it was the opinion of

most observers that the cyst itself originates in the remains of fœtal structures which occur in the locality in question —especially the organ of Giraldès and the duct of Müller— and that, growing amongst, and pressing upon, the vasa efferentia, it subsequently comes to communicate with one of the latter. This theory, however, is now generally believed to be unfounded, and it is considered that encysted hydroceles of the epididymis originate by dilatation of one of the ducts of the rete testis or of the coni vasculosi.

In addition to the encysted hydroceles above described,

FIG. 134.

An Encysted Hydrocele of the Epididymis. The testis is below the cyst.

small cysts at the caput epididymis are not uncommon. They have recently been very clearly classified by Dr. Joseph Griffiths in the *Journal of Anatomy and Physiology*. He recognises three varieties—(1) small sessile or pedunculated cysts containing clear fluid, not larger than a pea, and always multiple, situated in the cellular tissue of the caput epididymis, and originating as outgrowths or buds from the seminal tubes; (2) small, multiple cysts with fluid containing spermatozoa, originating in dilatation of the tubes of the coni vasculosi; (3) rather larger multiple

cysts, also containing spermatozoa, and originating in dilatation of the ducts of the rete testis. All these varieties are of such common occurrence at or after middle age, that they might almost be considered of physiological rather than pathological interest, and they are so minute that surgically they are of no importance.

Encysted hydrocele of the testis is extremely rare. It is situated in front of the testis, either between the tunica albuginea and the posterior layer of the tunica vaginalis, or else in the substance of the tunica albuginea itself. This form of encysted hydrocele is believed to originate in an injury which causes hæmorrhage, the extravasated blood being subsequently encysted. Small cysts with clear serous fluid are also occasionally seen on the surface of the tunica albuginea, and probably originate in dilatation of lymph spaces.

Encysted hydrocele of the spermatic cord is common, and is formed by distension of an unobliterated portion of that part of the tunica vaginalis which lies between the testis and the internal abdominal ring. The fluid is clear and serous, and does not contain spermatozoa. The sac is merely a small portion of peritoneum, and is usually not larger than a walnut. It may occupy any portion of the cord, but is most common just below the external ring. When in the inguinal canal, it may simulate an incomplete hernia.

Diffused hydrocele of the spermatic cord is a misleading name applied to a general infiltration of the cellular tissue of the cord with fluid—a condition for which a better and simpler name is œdema. It is of very rare occurrence.

Hydrocele of a hernial sac is a distension of a hernial sac with fluid secreted by the peritoneum of which the latter is composed. It occurs only when the hernia, as such, has been cured, the aperture of communication between the sac and the peritoneal cavity having been obliterated by adhesions or by omentum.

HÆMATOCELE.

A hæmatocele is a collection of blood either in the cavity of the tunica vaginalis or in a separate cyst in connection with the testis or the spermatic cord.

Vaginal hæmatocele, or hæmatocele of the tunica vaginalis, is by far the most common form, and is generally caused by some injury. In a few cases, however, no history of traumatism can be obtained, and some writers are of opinion that it is then of inflammatory origin, the blood

FIG. 135.

Section through a Hæmatocele. The cavity of the tunica vaginalis is filled with breaking down clot. The testis, which has been cut open, is quite healthy.

being supplied by exudation from newly developed vessels which vascularise the products of inflammation formed on the inner surface of the tunica vaginalis. Hæmatocele may also complicate new growths.

Hæmatocele may occur as an uncomplicated condition, but is much more often seen in connection with vaginal hydrocele. In the latter case, the effusion of blood may result from tapping, the trocar injuring one of the vessels ramifying over the distended serous membrane, or puncturing the testis. When there is no evidence of either

of these mishaps, the hæmorrhage is attributed to the giving way of one of the vessels on the tunica vaginalis as the result of the sudden withdrawal of pressure by evacuation of the hydrocele fluid. Independently of tapping or of hydrocele, a blow may result in effusion of blood and the formation of a hæmatocele.

The **fluid** in hæmatoceles varies much in quality and consistence. When the blood is mixed with hydrocele fluid, the colour is lighter in proportion to the amount of dilution. In recent cases not complicated by hydrocele, the hæmatocele contains pure blood. The longer the blood remains the more alteration does it undergo, though it is sometimes spontaneously absorbed.

If absorption does not occur, the red corpuscles break up, and their colouring matter becomes diffused, the serum is partially absorbed, and the remaining fluid becomes dark chocolate-brown, or black, and sometimes thick and syrupy.

The fibrinous constituents of the blood coagulate, and form clots, which, though at first soft and red, after a time become firm and of a yellowish or opaque-white colour. The most firm and fibrinous clot is always next to the serous membrane itself, and the more granular, friable, and darker coagulum is found in the centre of the hæmatocele. The clot is always most abundant on the parietal layer of the serous membrane; and the latter is, in addition, gradually thickened by the formation of fibrous tissue in its walls, to which after many years calcareous matter may be added. The testis is always healthy, though sometimes flattened by pressure.

Hæmatocele of the cord may be either diffused or encysted. The diffused hæmatocele is simply an extravasation of blood in the cellular tissue of the part, and the encysted variety is nothing more than an encysted hydrocele of the cord into which hæmorrhage has occurred. Both are rare.

Encysted hæmatocele of the testis originates in an effusion of blood beneath the tunica albuginea, whilst the

name of parenchymatous hæmatocele has been applied to effusions of blood in the substance of the gland itself resulting from injury.

VARICOCELE.

A varicocele is a varicose enlargement of the veins of the pampiniform plexus, and is of exceedingly common occurrence. It originates usually about puberty, and is more frequent in flabby, feeble, and lethargic subjects than in the strong and vigorous.

The frequency with which the spermatic veins are the seat of varicose enlargement is to be explained by their pendulous position, their want of muscular support, their length, and the paucity of valves. Varicocele is more common on the left side than on the right—a clinical fact which is partly accounted for by the somewhat greater length of the left spermatic vein, but is also supposed to be induced by the pressure of a loaded sigmoid flexure, and by the fact that the left spermatic vein opens into the renal vein at right angles to the blood-current, whilst the vein on the right side enters the cava obliquely, and empties itself in the direction of the blood-stream.

The affected veins show all the appearances typical of varicosity, being elongated, tortuous, dilated, and pouched. In some cases the distension affects the venules in the hilum testis, and in other cases the disease is complicated by thrombosis or phlebitis.

The testis on the affected side, if one side only be involved, is sometimes softer and a little smaller than its fellow. There is no reason to believe that the pressure of a varicocele ever causes atrophy of the testis, but it is probable that, when the varicose condition appears before puberty, it may interfere with the proper development of the gland.

CHAPTER LIII.

DISEASES OF THE FEMALE ORGANS OF GENERATION.

THE VULVA AND VAGINA.

Congenital deformities of these parts are in their slighter degrees by no means uncommon. The **hymen**, instead of being a crescentic fold of membrane, or a diaphragm with a central perforation, sometimes forms a **complete septum** across the vaginal orifice. Until puberty is reached, such a condition often passes unnoticed, but when menstruation commences the menstrual fluids are necessarily retained. If the hymen is slight and frail, it may give way before the pressure of the retained matter; more often, however, it is stretched, and bulged outwards through the labia, forming a cystic swelling, which in some cases, by compressing the urethra, interferes with the passage of urine.

Division of the vagina by a longitudinal septum into two cavities is a rarer malformation, and is usually associated with a bicornuate or double uterus. Occasionally the vagina is absent, and in most of these cases the uterus and ovaries are also undeveloped.

Hypertrophy of the labia or **clitoris** is in some cases the result of elephantiasis; in others, of chronic œdema or inflammation, but is sometimes apparently idiopathic.

Inflammation of the vagina has already been mentioned in connection with gonorrhœa, and noma vulvæ has been

included in the chapter on Gangrene. Vulvar abscesses, however, occur independently of venereal disease, and apparently originate in many cases in the sebaceous glands which are found in this situation. Warty growths and condylomata are generally the result of irritating discharges from the vagina, and, though often venereal, are not necessarily so.

Cystic tumours of the labia are sometimes of sebaceous origin, but in other cases originate from contusions or compression during labour. Such cysts come under the head of "hæmatoma," and contain dark, thick blood; they sometimes attain a very considerable size.

Innocent tumours of a more solid nature are usually **soft fibromata**. They grow slowly, are generally pendulous and pedunculated, and so soft as to give to the touch the feeling of fat or fluid.

The most common malignant growth of the labia is **epithelioma**, and in this situation such tumours grow with considerable rapidity. They often extend locally to the bladder or rectum, and cause secondary growths in the inguinal and pelvic glands, and more rarely in the viscera.

TUMOURS OF THE UTERUS.

Fibro-myoma or **uterine fibroid** is the most common new growth of the uterus. There are three chief varieties, named according to their respective relations to the tissues forming the uterine wall—(*a*) sub-peritoneal; (*b*) intramural or interstitial; (*c*) submucous.

The **sub-peritoneal** tumours usually spring from the fundus, and grow—covered only by peritoneum and sometimes by a thin layer of uterine tissue—towards the peritoneal cavity. They are more slow in their growth than the other varieties mentioned, and often cause no symptoms at all. Occasionally, however, they induce slight peritoneal effusion, and may contract adhesions to the intestines, ovaries, &c.

The **intramural** tumours grow in the substance of the uterine wall, with which the softer varieties appear to be directly continuous, the harder tumours being often completely encapsuled. They frequently cause considerable hypertrophy of the uterus, induce severe metrorrhagia, and are often multiple.

Fig. 136.

An Intramural Fibroid.

The **submucous** growths extend towards the uterine cavity, and tend to become pendulous or pedunculated. Like the intramural tumours, they cause uterine enlargement and hæmorrhage, and, in addition, by their pressure may cause sloughing of the mucous membrane which covers them. In such cases the growth itself may slough and be cast off.

Fibro-myomata of all kinds appear to owe their origin to anything which causes prolonged congestion or irritation of the uterus, and occur especially in women who have not passed the climacteric. During the involution of the organ after pregnancy, these growths not uncommonly undergo spontaneous absorption. After the menopause, not only do they usually cease to grow, but they also share in the atrophic changes which are in progress in the uterus after this period of life.

Fibro-myomata differ somewhat from one another in their naked-eye appearance, for, the more muscular tissue they have the more soft, red, and fleshy is their cut surface; whilst the more fibrous they are the whiter and denser are they on section. They are also liable to undergo various degenerative changes. In old people they are always very tough and fibrous, and in many become converted into calcareous masses. Less commonly, cysts are formed in their interiors and occasionally attain a great size.

On microscopical examination, a fibro-myoma, as its name implies, is found to be composed of a mixture of fibrous tissue and involuntary muscle fibre, these being combined in varying proportions.

Mucous polypi commonly grow from the mucous lining of the cervix or os. They are usually multiple, very vascular, and consist of simple pedunculated outgrowths of the mucous membranes from which they spring.

Malignant tumours of the uterus may be either carcinomata or sarcomata, and of these the former appear to be by far the most common.

Carcinoma may be columnar-celled, spheroidal-celled, or of the epitheliomatous or squamous-celled variety, the columnar-celled growth being the most common. These tumours possess the same microscopical structure as similar tumours in other parts of the body. According to the quantity of their fibrous stroma, they are either dense and scirrhous, or soft and medullary. They commonly originate in the cervix, but may spring from the fundus of the uterus.

Extending towards the uterine cavity on the one hand, they cause a foul discharge, with much hæmorrhage, and, growing towards the peritoneum on the other, they infiltrate the uterine ligaments, extend to the bladder or rectum and cause fistulous communications, contract adhesions to the abdominal organs, or induce acute peritonitis. They commonly cause glandular infection, and may become disseminated in the viscera. **Epithelioma** is usually described as occurring in two varieties—one originating usually in the mucous lining of the cervix, accompanied by but little warty growth, causing a general infiltration of the uterine tissue, and ulcerating at an early stage; the other commencing at the os uteri, beginning as a papillomatous, warty, or cauliflower growth, and often attaining a considerable size before ulceration begins. In either case extension to the neighbouring viscera, with foul discharge and glandular affection, is the usual sequel.

Sarcomata of the uterus have not been yet sufficiently observed to allow of any general rules being formulated as to their mode of growth or natural history.

TUMOURS OF THE OVARIES.

Ovarian tumours are either cystic or solid, and many of them are cystic with solid growths.

Ovarian cystomata appear to be developed from Graafian vesicles, and in some cases perhaps from corpora lutea. The cysts may be either single or multiple, and in the latter case the septa dividing them from each other are liable to become so thin as to allow of communication between the cavities. The fluid contained in the cysts varies much. It is always albuminous, and in the single cysts is frequently thin, watery, and yellowish. In the multiple cysts it is more likely to be thick and opaque, sometimes acquiring the consistence of glue and a dark-brown colour. It occasionally contains cholesterin.

In the case of multiple cysts there is often solid growth extending into the cystic cavities. Such tumours are

named **proliferous cysts**. The solid ingrowth is generally soft and myxomatous, and is itself liable to degenerate and form secondary cysts filled with blood-stained fluid. In rarer instances the solid growth is sarcomatous or carcinomatous. All varieties of cysts are liable to inflame, and to contract adhesions to the abdominal viscera and parietes.

Dermoid cysts are also met with in the ovary. Like similar tumours in other parts, they are liable, after long periods of quiescence, to take on active growth. They contain sebaceous matter, hair, bones, teeth, &c., and sometimes attain considerable size.

Parovarian cysts, or cysts of the broad ligament, are situated in the latter structure, and not in the ovary itself. They are single, contain a clear, watery fluid, and never contain solid ingrowths.

Solid fibrous and malignant tumours of the ovary are also met with, but are not of common occurrence.

CHAPTER LIV.

DISEASES OF THE BREAST.

Diseases of the breast are infinitely more common in women than in men, the mammary gland in the latter being in a rudimentary state. It must be understood, therefore, that the following description of diseases of the breast refers, unless the contrary be expressly stated, solely to the female breast.

INFLAMMATION AND ABSCESS.

The hyperæmia and irritation produced by suckling are the most common causes of **acute** inflammation of the breast. Most cases occur either soon after pregnancy or when suckling has been unduly prolonged to some ten or twelve months or more.

In the slighter cases the nipple and areola alone are affected, the skin covering them becoming at first red and swollen, and afterwards eczematous, excoriated, and raw. Cracks or fissures also form, and definite ulcers may be developed. Sometimes the inflammation terminates in the formation of a small superficial abscess in the areola or neighbouring subcutaneous tissue.

In another class of cases the gland tissue itself is involved in the inflammatory process, which appears, in some cases at least, to extend from a cracked and fissured nipple. A single lobule only is at first implicated, and in its substance pus may collect, whilst the rest of the gland remains unaffected. If not treated, however, by early and free

incision, the pus soon makes its way to other lobules, and before it obtains an exit through the skin, becomes more or less diffused. In consequence of the fascial prolongations of the gland capsule, a single opening now will not suffice to give ready exit to the matter, and incisions have frequently to be made into each of the lobules implicated in the suppuration.

The cellular tissue behind the breast is less frequently the seat of suppuration than the nipple or the gland itself. When pus forms in this situation, it pushes the whole breast forwards and makes its way to the surface at the lower margin of the mamma.

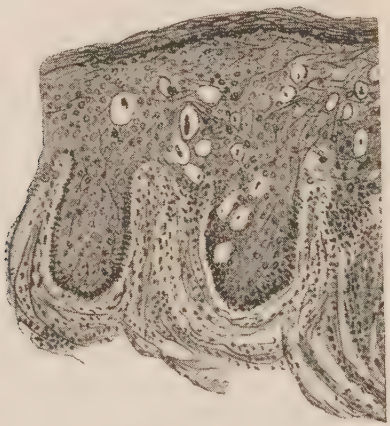
Chronic abscess may also develop in the breast, and like the more acute inflammation, is generally the sequel of parturition or of a miscarriage. The pus is often surrounded by a considerable amount of fibrous tissue, and, if it be placed in the deeper parts of the gland, may closely simulate a solid growth.

Chronic interstitial mastitis is a form of inflammation of the breast which is seen most commonly in women who have passed the climacteric. It is generally confined to one or two lobules of the gland, and is characterised by the formation of much fibrous tissue, with consequent induration, thickening, and nodulation of the mammary substance. On section, the affected lobules are found to be more white, fibrous, and dense than the remainder of the breast, whilst a microscopical examination shows infiltration with leucocytes, formation of fibrous tissue, fatty degeneration and destruction of the epithelium, and slight dilatation of the ducts and acini in the form of minute cysts. This condition not only simulates scirrhous cancer, but is occasionally the precursor of a carcinomatous growth.

Paget's disease of the nipple is the name given to a peculiar form of eczema limited at first to the nipple and areola, and characterised by its obstinate resistance to all treatment, its tendency to ulcerate, and its liability to be succeeded by the development of carcinoma.

The areola is at first bright red and inflamed, the epithelium subsequently peeling off in branny scales and leaving a raw surface, from which exudes a watery discharge, which tends to dry and form scales. From the commencement of the disease the affected parts are much more red and congested than are eczematous tissues. After a time, ulceration succeeds, extending to, and destroying the nipple in many cases.

FIG. 137.



Skin from "Paget's Disease" of the Nipple, showing the large vacuolated cells in the epidermis.

The nature of the process has been carefully described by Mr. Butlin; and in thirteen breasts which I have myself examined I have found very nearly the same conditions that were seen by him.

The process originates in an inflammation of the derma, with small cell exudation beneath the epithelium. The latter, in its turn, is implicated, its cells being loosened from one another by fluid exudation, and finally cast off. The derma being exposed, the inflammatory process advances more rapidly, and proceeds to the formation of pus and the destruction of the true skin. The inflammation now extends along the ducts, causing their epithelial lining

to proliferate, the cells sometimes collecting in masses, which fill the tubes. Following this, there is a tendency for the epithelial cells in the acini or ducts to grow out into the surrounding tissue and to take on cancerous growth, an event which is generally preceded by inflammatory changes in the connective-tissue stroma of the mamma, indicated by exudation of leucocytes and fibrous thickening. The cancerous growth is sometimes directly continuous with the nipple, but is more often quite separate from it and placed deep in the breast. It is usually a spheroidal-celled scirrhous carcinoma, though columnar-celled tumours have been described.

A microscopical examination shows in the epidermis numerous round or oval vacuolated cells, which are most numerous in the superficial layers of the epithelium. The appearances presented by these cells are characteristic of the disease.

Paget's disease must not be confused with an eczematous condition of the nipple which is sometimes seen as a *sequel* to a cancerous growth, and which appears to result from some irritating discharge from the ducts.

Hypertrophy of the breasts, in the true sense of the term, is very rare, but cases have been recorded in which the gland has attained many pounds weight, and has, on account of its weight, required amputation. Most hypertrophied breasts owe much of their size to the presence of numerous fibro-adenomatous tumours.

Galactocele is the name given to a cyst of the breast containing milk. It develops in connection with lactation, and is supposed to originate in rupture of one of the milk-ducts. Such a cyst may contain a pint or more of fluid, which in some cases is entirely absorbed. In other cases the fluid is reduced to a caseous pulp, occasionally gives rise to suppuration, and is discharged together with the pus.

CHAPTER LV.

TUMOURS OF THE BREAST.

Tumours of the breast are commonly of a mixed nature, containing both connective-tissue and epithelial elements. It is not, therefore, possible to classify them under either the connective-tissue or epithelial new growths. The classification of these tumours is made still more difficult by the fact that they are often complicated by cysts; nevertheless, their structure is generally perfectly clear.

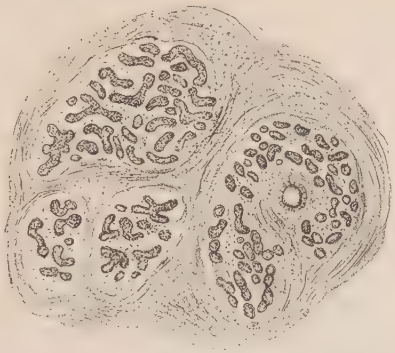
ADENOMA, ADENO-FIBROMA, ADENO-SARCOMA.

These are the names given to a class of mammary tumours which all contain a certain amount of breast tissue embedded in a fibrous or fibro-sarcomatous matrix. The pure **adenoma** is very rare if the name is strictly limited to tumours composed throughout of tissue such as is found in a lobe of the normal mamma. I have seen only two examples amongst a large number of mammary growths. They were yellowish-white in colour, soft, lobulated, and encapsuled, and, on section, showed, under the microscope, numerous ducts and acini embedded in a fibrous matrix. The only point in which their structure differed from that of a normal breast was that the gland tissue was irregular in the manner of its arrangement.

Adeno-fibromata are the growths which are commonly spoken of as "chronic mammary tumours," although this name is used very loosely, and is also applied to many of the adeno-sarcomata as well. The adeno-fibroma is the

commonest of breast tumours. It is generally single, movable in the mamma, lobulated on the surface, encap-

FIG. 138.



Adenoma of the Breast. The tumour does not materially differ from normal breast tissue. (Zeiss, A.)

suled except in its deeper parts, where it is commonly continuous with the surrounding gland; on section, it is found to be firm, white, and fibrous, and to exude a little

FIG. 139.

Fibro-adenoma of the Breast. The glandular tissue is badly developed, and is mingled with much fibrous tissue. (Zeiss, A.)

viscid fluid. Microscopical examination shows a stroma of fibrous tissue, in which, in typical specimens, but few con-

nective-tissue cells are found, mixed with a variable amount of gland tissue. In some cases the gland tissue is exceedingly scanty, not more than one or two acini appearing in a single field of the microscope; in others, as much as a quarter or a third of the growth is composed of glandular elements. The acini and ducts are usually not well formed, the lumen of the tubes, as well as their outline and limiting membrane, being ill-defined, and their epithelial cells small and more numerous than natural. (See Fig. 139.)

Adeno-sarcomata are not separated by any hard-and-fast line from the adeno-fibromata, there being all grades of intermediate growth. An adeno-sarcoma resembles an adeno-fibroma in its encapsulation and its lobulated surface, but is softer, less fibrous, and more gelatinous on section. Microscopical examination shows a stroma composed of delicate fibrous tissue, containing numerous oval or spindle cells mingled with a good deal of homogeneous mucoid material and a few branched or stellate cells. In the midst of this complex stroma is embedded gland tissue, such as has already been described as occurring in the adeno-fibromata.

CYSTIC ADENO-FIBROMA AND CYSTIC ADENO-SARCOMA. — SERO-CYSTIC TUMOURS. — PROLIFEROUS CYSTS.

In many of the adeno-fibromata and adeno-sarcomata, cysts develop from the mammary acini and ducts. These vary greatly in size in different tumours and in different parts of the same growth. In many tumours which to the naked eye are apparently not cystic, the microscope shows that here and there throughout the sections the acini and ducts are distended, and form irregular cavities or spaces, in some places elongated and slit-like, at other parts more rounded or oval. In other tumours, cysts of all sizes, some a couple of inches in diameter, are found, filled with serous or mucoid fluid, and containing in most cases a certain amount of solid growth. The cysts are

lined by one or more layers of short columnar or spheroidal epithelium, though in many cases where the cavities are large the epithelial lining is very liable to separate during the manipulation necessary for the preparation of a microscopical section.

Cystic adeno-fibromata and adeno-sarcomata have long been known as the "sero-cystic tumours of Brodie," and under that heading growths of very different structures have been classified. These tumours are generally of con-

FIG. 140.

Fibro-adenoma of the Breast with Cysts. The cysts are developed from the mammary acini, and are lined by epithelium. (Zeiss, A.)

siderable size, and may attain several pounds in weight. Their surface is bossed, the cysts presenting themselves as rounded swellings of various dimensions. Their consistence is unequal; elastic or fluctuating in some parts, they are solid in others. A section liberates a certain amount of clear or slightly blood-stained serous or mucous fluid, and shows that the tumour as a whole is encapsuled. The solid portions of the growth are either white and fibrous, or pinkish, fleshy, soft, mucoid, or gelatinous. The cysts in some cases are chiefly filled with fluid, in others they contain masses of new growth, and are then named

"proliferous." The solid growth, however, is not really within the cyst cavity, although to the naked eye it may appear to be so. It is simply a part of the new growth, forming the bulk of the tumour, which has invaginated the cyst-wall in front of it, and thrust it into its own cavity. The protruding mass is always covered by the epithelial

FIG. 141.

Fibro-sarcoma of the Breast with Cysts (Sero-cystic Disease). The cysts are lined by epithelium. The new growth springs from the connective tissue of the mamma, and several processes of it have extended into the cavity of the large central cyst, and partly filled the latter. These intra-cystic growths are covered by the epithelium lining the cyst. (Zeiss, A.)

cells which line the cysts, and does not penetrate or fungate through them.

Microscopical examination shows that in the cystic adeno-fibromata the growths have the same structure as the adeno-fibromata, which are uncomplicated with cysts; similarly, the cystic adeno-sarcomata have that of the adeno-sarcomata without cysts. Many of the so-called "chronic mammary tumours" are simply proliferous cystic tumours on a small scale.

The clinical course run by the tumours above

mentioned differs according to their structure. The solid growths, whose structure is chiefly fibrous and glandular—the adeno-fibromata—develop in young women, and are most common between the ages of seventeen and twenty-seven. They grow slowly, and never attain a great bulk, being seldom larger than a walnut. They do not affect the lymphatic glands or implicate the skin, and are generally freely movable in the breast. In most cases they do not cause severe pain, and are not tender, though some few of them are so painful as to have acquired the name of "painful mammary tumours." These do not differ in their structure from the painless growths.

The adeno-sarcomata grow more rapidly than the adeno-fibromata, and attain a greater size. They are softer than the latter, but do not implicate the tissue outside the breast or involve the lymphatic glands. Clinically as well as structurally they are not definitely separated from the more fibrous growths.

The proliferous cystic tumours are generally seen in women somewhat older than those in whom the solid growths usually occur, but, like the latter, their clinical course depends on their structure. If the intra-cystic growths and the stroma of the tumour be fibrous, several years may elapse before the patient applies for relief, and I have seen breasts removed in which the swelling had existed as long as six or seven years. When, however, the stroma is sarcomatous the rapidity of increase is vastly greater, and is in proportion to the abundance of the cell-growth and the absence of fully formed fibrous tissue. The breast in bad cases may double its size within six or eight months. The more sarcomatous the tumour, the more likely it is to implicate the skin, which in these cases is destroyed rather by pressure than by infiltration, and allows the protrusion of a bleeding mass which soon ulcerates from exposure, and discharges pus mixed with blood-stained fluid from the cysts.

Before the skin is involved, a breast containing a pro-

liferous cystic growth presents a very irregular outline, the surface being raised so as to form rounded swellings of various sizes which correspond to the cysts above described. A serous blood-stained discharge is often noticed to exude from the nipple, and can be increased by pressure. This results from a communication existing between one of the milk ducts and a cyst containing fluid. However large a size the proliferous growths acquire, they practically never implicate the lymphatic glands, and only a few of the more sarcomatous ones become disseminated.

Fibrous tumours unmixed with any gland tissue are rare, and do not differ materially from the adeno-fibromata.

Sarcomatous growths, solid throughout, and containing no gland tissue, are more common than the pure fibromata, but are still of comparatively rare occurrence. Round, oval and spindle-celled sarcomata have been seen in the breast, and here, as elsewhere, the round-celled growths develop more rapidly and show a greater tendency to disseminate than do the spindle-celled tumours. The axillary glands are very rarely involved.

Cartilaginous and **bony** tumours of the breast are very uncommon, there being only about half a dozen cases on record. In several of these also the cartilage or bone was merely a part of a growth which was otherwise sarcomatous.

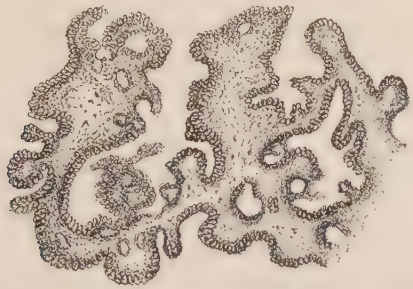
Cysts of the breast are usually divided into retention cysts and serous cysts.

Retention cysts are formed by distension of the mammary acini and ducts. They may be either single or multiple, but the latter is the more common form. They are frequently seen in elderly women in whom the breasts are undergoing atrophic changes, and are then sometimes called "involution cysts," but they may occur at any period of life. Their size varies much; in some cases they are quite microscopic, in others they contain five or six ounces of fluid. The latter is in some cysts quite clear and watery, in others blood-stained, thick, and viscid. The tension

inside the cyst is occasionally so great that when the latter is situated in the deeper parts of the mamma it simulates a solid tumour.

Microscopical examination of different specimens shows all stages of development from the mammary tissue, the cyst-wall being formed of fibrous tissue lined by epithelium, which is either spheroidal or columnar according as the cyst has developed from an acinus or a duct. The larger the cyst the more flattened is its epithelial lining, and the greater tendency is there for the latter either to become quite flat or to be shed. Thus it is common to find cysts

FIG. 142.



Portion of a Papillomatous Ingrowth from a Duct-papilloma of the Breast. It consists of a delicate stroma of connective tissue covered by columnar epithelial cells. (Zeiss, A.)

which are lined in part by flattened epithelium, which in the rest of the circumference is absent.

In some cases **papillary** growths of a bright red colour, like minute raspberries, grow from the cyst-walls. They are composed of a very delicate connective tissue with thin-walled blood-vessels covered by epithelium, and are very liable to bleed.

In cases of cysts containing these papillæ, I have seen considerable discharge of blood-stained fluid from the nipple. They grow especially in those cysts which are formed by distension of the ducts. (See Fig. 142.)

Serous cysts of the breast are supposed to be formed

by distension of a connective-tissue space or lymph space with serous fluid. There is really no proof that cysts are formed in this way in the breast, and it has never been explained why such cysts should be so common when they are associated with the connective tissue of the mamma, whilst in other parts of the body they are only very rarely found as new formations. I myself am of the opinion that the so-called serous cysts are really formed by the distension of acini, and that the failure to find an epithelial lining results either from the cells of the latter having become extremely flattened, so as to resemble endothelium, or having been shed as already described. My reason for arriving at this conclusion is, that an examination of many cystic breasts has shown me all stages between cysts lined by typical glandular epithelium, and those in which no such lining could be demonstrated.

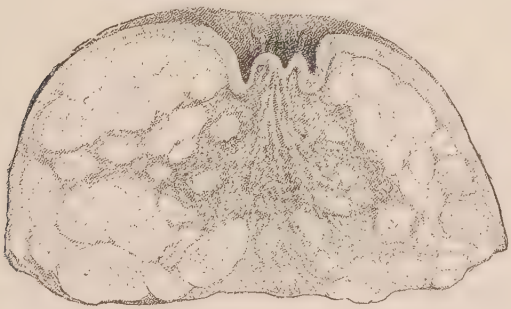
CARCINOMA.

Spheroidal-celled carcinoma is met with in the breast in all its varieties—scirrhous, encephaloid, and colloid, but of these the first is infinitely the most common.

Scirrhous carcinoma is most often observed in women over forty, but is occasionally seen in patients younger than this by some eight or ten years or more. It usually commences as a hard rounded lump, which is movable and painless—conditions which are very commonly considered to be incompatible with malignancy, but it is therefore all the more necessary to bear them in mind. As the lump increases, it becomes rough, irregular, and nodular, more evidently hard, and less movable. When the tumour approaches the skin, it commonly causes the latter to become dimpled or puckered, and at a later date widely adherent. At the same time, or sooner, the nipple is commonly retracted, and round hard nodules, the size of a pea, or smaller, may develop in the derma. The affected skin soon becomes red, shiny, and often œdematous, finally ulcerating at one spot, and allowing the protrusion of a

bleeding mass of cancer. The tumour, however, extends not only towards the surface, but also to the deeper parts, infiltrating the pectoral muscles, fixing the breast so that it becomes perfectly immovable, and even extending down to the ribs themselves. At an early period the lymphatic glands in the axilla become enlarged and hard, the glands above the clavicle following suit as the disease progresses. The masses formed by the cancerous lymphatics sometimes attain a great size, and, by infiltrating the axillary nerves, may cause the patient intense pain. Œdema of the arm is often produced by their pressure on the main vein.

FIG. 143.



Section of a Breast with Scirrhous Carcinoma. The growth infiltrates the fat, and has caused retraction of the nipple. (From a specimen in St. Thomas's Hospital Museum.)

By the time the skin has given way, and the axillary glands are enlarged the patient's health also fails, emaciation being sometimes very rapid. Death may result from exhaustion caused by pain and discharge of blood and pus from the ulcerating mass, but it is often the result of other complications. One of the commonest of these is pleurisy, a condition very readily accounted for when we consider that, by the time that the tumour has penetrated the pectoral muscle, but little intervenes between it and the pleura. The latter may be involved either by direct extension of the growth, by infection through the

lymphatics, or by secondary deposit. A fatal termination may also result from secondary growths in internal organs. The viscera most commonly affected are the liver and lungs, but I have myself seen cases in which all the viscera in the abdomen and thorax were involved at once. Growths in the bones also are by no means uncommon, and are frequently very insidious, perhaps causing no symptoms of any kind until some slight movement results in a spontaneous fracture.

The above description of the course of a scirrhous cancer applies to most cases, but not to all, and the exceptions demand a brief recognition. Instead of commencing as an isolated small lump, the tumour may from the beginning affect a large portion of the whole breast, being diffused as it were throughout its substance, and presenting to the touch an ill-defined, indurated swelling. In such cases especially, the skin is liable to be involved over a large area, being at first puckered and lumpy, but afterwards converted into a brawny mass of leathery consistence, and of a dusky-red tint, which envelops the thorax in a rigid inelastic sheath, which has been compared to a hide or a cuirass.

Atrophic scirrhus is the term applied to a form of hard carcinoma, which occurs in elderly and thin women, and is characterised by the constant tendency of the tumour to shrink and contract rather than to increase in size and fungate. Growths such as this are very chronic, and may last several years without making notable progress. They may, nevertheless, destroy life by causing secondary growths in the viscera.

On section, scirrhous carcinoma of the breast grates or creaks under the knife. Its cut surface, which has been compared to that of a raw potato or an unripe pear, is concave, greyish or bluish-grey in tint, and marked by irregular white dots and streaks. By a little pressure some of this white matter may be squeezed out, and is found, on microscopical examination, to consist of broken-down epithelial cells. Scraping with a knife generally produces

a few drops of dirty opalescent fluid, also containing epithelial cells of all shapes and sizes. The margin of the tumour is very irregular and ill-defined, close examination often showing that, as it implicates the neighbouring fat or gland tissue, portions of the latter are, as it were, surrounded by offshoots from the main growth preparatory to their absorption: small pieces of fat may thus be seen embedded in the growing edge. There is never any trace of a capsule. The retraction of the skin or of the nipple is produced by the contraction of the fibrous stroma of the tumour. The nipple is drawn down by its main ducts becoming involved, and consequently may entirely escape retraction if the tumour is limited to the margin of the mamma. In some cases cystic cavities form in the more central portions of the mass. They result from degenerative changes, and contain a dark blood-stained fluid. More rarely, suppuration occurs, and in exceptional cases there is a considerable collection of pus. Sections of the lymphatic glands, and of other secondary growths, show appearances similar to those presented by the original tumour.

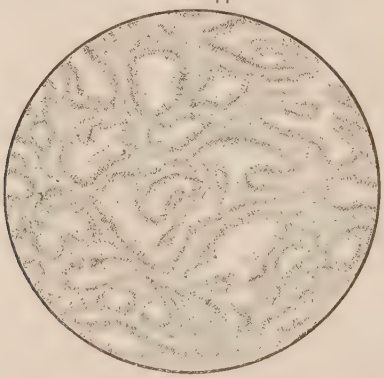
Microscopical examination shows the structure common to all the scirrhous carcinomata. The white masses which can be squeezed out are formed by degeneration of the cell masses, as well as by a catarrh of the ducts, the epithelium from which is unable to escape owing to the pressure of the growth.

VILLOUS OR DUCT CANCER.

Villous cancer is a form of carcinoma which is much softer than scirrhous carcinoma, and either of a red tint, or else so filled with extravasated blood as to be mistaken for a melanotic tumour, or a blood-cyst. Closer examination reveals the presence of minute cysts, filled with blood-stained fluid, and some soft solid growth. The microscope shows a fibrous stroma enclosing alveoli of considerable size. These alveoli are identical with the cysts seen by the naked eye, and are lined by columnar epithelium. From

their walls, "villous" or papillary processes sprout, formed of a delicate stroma of fibrous tissue enclosing blood-vessels,

FIG. 144.



Section of a Duct Cancer showing the interlacing processes covered by columnar epithelium. (Crouch, $\frac{2}{5}$ in.)

and covered by columnar epithelium. Both stroma and alveoli contain much extravasated blood.

FIG. 145.

Section of a Breast with nodules of duct cancer in it and in the mammary fat.

Tumours of this nature are not so rare as was previously thought. Their clinical course seems to differ from that of

scirrhous cancer. They grow more slowly, are not so liable to affect the glands, do not recur so quickly or frequently after removal, and show little tendency to disseminate. Their growth is often associated with a discharge of blood-stained serum from the nipple, but they do not produce that retraction of the nipple which so often accompanies scirrhous cancer.

ENCEPHALOID CANCER.

Encephaloid carcinoma of the breast is decidedly very rare—*i.e.*, if the term be limited, as it ought to be, to soft brain-like tumours of a dirty-white colour, which readily break down on section. Their growth is very rapid, and their tendency to disseminate great. Such tumours have already been fully described at p. 194.

COLLOID CANCER.

Colloid cancer of the breast is also rare, but not so much so as the encephaloid variety. Its clinical course differs from that of the ordinary scirrhous tumours in that the tumours are less liable to affect the glands and to disseminate, and if removed do not show so great a tendency to recur. Its structure has already been described at p. 195.

Tumours of the **male breast** are of rare occurrence. I have seen instances of spindle-celled sarcoma, of scirrhous and encephaloid carcinoma as well as of adeno-fibromata; but in all the growths the absence of mammary tissue, and consequently of cysts, is very marked.

CHAPTER LVI.

DISEASES OF THE LIPS, MOUTH, AND SALIVARY GLANDS.

The congenital deformities of the lips and palate are dealt with in another chapter, and it only remains to mention that in some cases there is an **hypertrophy of the mucous membrane**, causing a protrusion and thickening which may be very unsightly. Slight thickenings are comparatively common in strumous subjects, but, in addition to these, there are other cases in which the hypertrophy is much greater and more definite, and is attributed by Mr. Bryant to overgrowth of the submucous glands. The hypertrophy is usually most marked in the upper lip. Examples of this disease are by no means common.

In other cases of acquired deformity the aperture of the mouth is much narrowed by the contraction of cicatrices, the result of lupus or cancrum oris, and, in older subjects, of epithelioma or rodent ulcer.

Deep cracks and fissures sometimes form in the lower lip, especially in the middle line. At first they are quite superficial, but, if left untreated, they sometimes extend to a considerable depth, and even when healed leave a permanent depression. The fissures which are sometimes seen about the angles of the mouth in children are frequently indicative of congenital syphilis, but they occur also in strumous patients.

Ulcers of the mucous surface of the lips are of common occurrence in early life, and are usually due to errors in

diet, leading to dyspepsia. In patients, also, who are seriously ill from any cause, superficial ulcerations and excoriations are commonly seen. More rarely the ulceration extends deeply, and causes considerable destruction of the submucous tissues. It should be remembered that these ulcers are not merely of local origin, but are dependent rather on the constitutional condition of the patient.

Primary syphilitic sores are of sufficiently frequent occurrence on the lips to merit special mention. They are generally said to be most common on the upper lip, and are usually met with in comparatively young patients. They frequently afford excellent examples of the true Hunterian chancre, being raised, definitely circumscribed, with a raw excoriated surface, and a very indurated base. The lymphatic glands are early affected, not only those in the sub-maxillary regions, but those over the ramus of the jaw and in the anterior triangles of the neck being implicated. These glands attain a much greater size, and are much more painful and inflamed than are the inguinal glands in the case of an infecting sore on the penis. The sores in question are generally attributed to contact with another person suffering from some secondary syphilitic ulceration about the lips or tongue, and are followed by the usual evidences of constitutional syphilis.

TUMOURS OF THE LIPS.

Innocent tumours of the lips are not of very common occurrence, but several varieties are to be met with. **Cysts** of the mucous surface are amongst the most common. They are seldom larger than a hazel-nut, and contain a clear viscid fluid; they are formed by retention of the secretion in one of the mucous glands.

Nævoid growths for the most part present no very definite peculiarities which require special mention in this place; they have already been described in the chapters on Tumours. The lips are, however, common sites for their development, and here more than elsewhere large blood-

cysts are apt to form, presenting themselves as purple swellings, rounded in shape, and sometimes attaining considerable size.

Glandular tumours of the lips, of slow growth, lobulated, and yellowish-white on section, have been described by Sir James Paget, under the name of labial glandular tumours; they appear to be of rare occurrence.

Papillomata or warty growths are not common. They present the characters shown by such growths in other situations, being raised, with roughened surfaces covered by thickened epithelium.

Epithelioma is the commonest tumour of the lips, but is infinitely more common on the lower lip than the upper. The subjects of such growths are much more often men than women, and are usually past middle life. The tumour commences, as a rule, at or near the line of junction of the skin with the mucous membrane, and attacks by preference that part of the lower lip which is close to the angle. Its earliest appearance is in the form of a small papule or wart; as this increases in size the superficial and most central part becomes excoriated and sore, and after a time definitely ulcerated. The ulcer, once formed, never attempts to heal, its surface is sloughy, and its discharge thin and watery, it presents no appearance of granulations. If the lip beneath the seat of growth be felt between the finger and thumb, it will be found that the tumour has not only grown towards the surface, but has also infiltrated the subjacent tissues, and the latter, in consequence, feel firm and indurated. If no treatment be adopted, the growth extends to the neighbouring parts, and may thus implicate the jaw, and extend into the tissues forming the floor of the mouth. Meanwhile, the lymphatic glands have become the seat of secondary growth, which infiltrates and destroys the surrounding tissues, very soon causing the glands to become fixed, and after a time breaking down and forming a fungating mass, which protrudes through the skin. Death finally ensues from exhaustion induced by the pain, dis-

charge, and difficulty in swallowing. If an epithelioma be freely removed in the early stage, such complications may be entirely avoided, and although recurrence is common, it is by no means inevitable, whilst dissemination is decidedly rare.

DISEASES OF THE PALATE.

The soft palate is not often the seat of simple inflammation, except in connection with catarrh of the neighbouring mucous surfaces; follicular ulcers, due to dyspepsia, do, however, form on it at the same time that they affect the tongue. The palate, soft or hard, may be attacked by either strumous or syphilitic ulceration, and in each case perforation may result. In some instances there is considerable caries or necrosis of the palatine processes, but such extensive mischief is more common in connection with syphilis than with struma. Perforations of large size are very liable to be permanent, but the smaller apertures not infrequently close.

Tumours of the palate are not very rare. The two chief forms of growth are sarcoma and fibro-adenoma. The sarcomata are usually of the round- or oval-celled variety, of rapid growth, occasionally affecting the lymphatic glands, and prone to recur after removal. They present as rounded, smooth, highly elastic swellings, usually limited to one side of the palatine arch, often in part encapsuled, but at other times infiltrating the tissues amongst which they lie. The fibro-adenomata are of much slower growth, and I have seen one which at the end of twelve years was little larger than a walnut. They resemble the sarcomata in their clinical appearance, but are more definitely encapsuled, and do not affect the glands or recur after removal. On section, they closely resemble the so-called "parotid glandular tumours," being friable, granular, and soft. Microscopically examined, they have a fibrous or homogeneous stroma, containing a varying quantity of gland tubes and irregularly arranged masses of epithelial cells. Epitheliomata and

mucous cysts, besides other and more uncommon forms of growth, are also met with on the palate.

DISEASES OF THE TONSILS.

The tonsils are very frequently attacked by inflammations, both acute and chronic. **Simple acute tonsillitis** most often results from exposure to cold. It is usually at first limited to one tonsil, but may afterwards affect that of the opposite side. The inflammation is liable to end in abscess, and the soft palate, the ary-epiglottic folds and surrounding parts, become much congested and œdematous. There is much pain, especially on swallowing, and often a considerable amount of fever with severe constitutional disturbance. Respiration is sometimes rendered a little difficult, but there is never urgent dyspnœa. After the abscess has burst relief is speedily obtained. The patients are most often young adults, and anything that impairs the general health appears to act as a predisposing cause of the affection. Many people are liable to frequent recurrences at intervals of a year or less, and in the opinion of some observers the disease is associated with the rheumatic diathesis.

Chronic tonsillitis is of very common occurrence in children, and is certainly most common in those of a strumous disposition. The consequence of chronic inflammation is an enlargement of the whole gland, with thickening of its mucous surface, and formation of thick, viscid, follicular secretion. Both tonsils are generally affected, and may increase to such a size that they meet in the middle line. Examined after removal they are generally found to be more tough and fibrous than natural, but are sometimes flabby and pendulous; the surface is deeply pitted. Microscopic examination shows an increase of the fibrous stroma, resulting in obstruction to the follicles and consequent retention of secretion and distension of the glandular structure. In chronic tonsillitis there are often adenoid growths and inflammation of the neighbouring mucous surfaces, causing thickening of the Eustachian tubes, with

deafness, and a nasal, unpleasant voice. In such cases respiration is sometimes interfered with to such an extent that the thoracic walls are driven inwards by the atmospheric pressure, and the chest becomes marked by the transverse groove so often seen in rickets.

Ulcerative tonsillitis is specially liable to be produced by exposure to bad smells, such as result from defective drainage, but is also met with in patients who are overworked and debilitated from any cause. The ulcers are multiple, but generally superficial, and heal readily under treatment. The affection of the tonsils in scarlatina is by no means always so trifling, and in some cases sloughing ensues, which may reach a most dangerous extent, and may produce alarming hæmorrhage by opening either the tonsillar arteries themselves, or, much more rarely, the internal carotid.

The ulceration of the tonsils in diphtheria is of the same nature as that which always characterises diphtheritic inflammations, and here also sloughing may occur to a dangerous extent. The affections of the tonsils in secondary syphilis have been already described.

Tumours of the tonsils are not common. Perhaps that most often seen is epithelioma, but lympho-sarcomatous growths and fibrous polypi also occur.

DISEASES OF THE PHARYNX.

Superficial **inflammation** of the mucous membrane of the pharynx, with enlargement of the lymphoid follicles and the formation of follicular ulcers, is tolerably common, and is frequently associated with abnormal conditions of the nares or tonsils. Inflammation going on to the formation of abscess is more uncommon, and may result either from mechanical injury caused by swallowing bones or other hard structures, or from suppuration commencing outside the pharynx proper. Abscesses having the latter origin are sometimes formed in connection with caries of the cervical vertebræ, but much more often result from tubercle in the deep lymphatic glands. In either case the

collection of pus readily separates the posterior pharyngeal wall from its loose connections with the muscles covering the front of the spinal column, and points as a **retro-pharyngeal** or **post-pharyngeal** abscess, sometimes giving rise to much dyspnœa and dysphagia.

The pharynx may be the seat of either **tubercular** or **syphilitic** ulceration. The former occurs especially in children; the latter, which is more common, in patients of more advanced age, with other evidences of syphilis. In either case the ulceration may be very extensive, and may result in much destruction of tissue; after the destructive process has ceased the trouble is by no means at an end, for during the process of cicatrisation the soft palate frequently contracts adhesions to the pharynx, and in bad cases may be so universally attached that the posterior nares become completely shut off from the pharynx. Such results as these are almost limited to the syphilitic variety of ulceration.

Tumours growing from the pharynx itself are rare, and the only varieties worth mentioning are soft fibromata, which are pendulous, and sometimes attain a considerable size. The naso-pharyngeal growths are described in the chapter on Diseases of the Nose.

DISEASES OF THE FLOOR OF THE MOUTH.

The floor of the mouth is not affected by any forms of inflammation apart from those which involve the lips, tonsils, or tongue, but **cysts** in this situation are not uncommon. Some of these are simple mucous cysts, like those which occur on the lips, but others are more deeply seated and considerably larger. One of the most common of the latter is that known by the name of **ranula**. This occurs on one side of the floor of the mouth in the form of a tense, shiny, bluish swelling, pushing the tongue upwards and towards the opposite cheek, and often causing a very considerable protrusion in the sub-maxillary region. When of larger size, a ranula passes more towards the

middle line of the mouth, and ceases to present the unilateral appearance above mentioned. When opened, the contents are found to be a thick, tenacious, clear fluid, resembling inspissated saliva; and, indeed, such cysts were formerly supposed to originate in an obstruction to one of the salivary ducts. This origin is no longer credited, for it can often be demonstrated that the ducts are quite free, and it is now supposed that a ranula originates in a dilatation of one of the main ducts of the two pairs of small mucous glands which are found on each side of the frænum linguæ, and are known as the glands of Nuhn and Blandin. The obstruction is probably of inflammatory origin, but may in some cases be mechanical, and due to the presence of foreign bodies. The suggestion that ranula originates in a mucous bursa on the upper surface of the genio-hyo-glossus muscle, is negatived by the fact that no such bursa has ever been demonstrated.

Another form of cyst which attains a considerable size in the floor of the mouth is the **dermoid cyst**, which is usually met with in the middle line between the genio-hyo-glossi muscles. Although of congenital origin, such cysts do not usually attain a sufficient size to attract attention before the age of fifteen or twenty, and in some cases the patients are still older when the swelling is first noticed. These cysts may be distinguished from ranulas by their central position, slow growth, and the considerable extent to which they project between the chin and the hyoid bone. They are probably due to imperfect obliteration of the lingual duct, and their walls are composed of skin, with hairs, sweat glands, and sebaceous glands in more or less abundance. They contain sebaceous matter, and, if they have been inflamed, are very firmly attached to the tissues amongst which they lie. They occasionally suppurate. Similar cysts are more rarely met with in the sub-maxillary region, and are in this situation apparently connected with the remains of one of the branchial clefts.

In addition to these innocent growths, the floor of the

mouth may be the seat of **epithelioma**, which runs the usual course of that disease when met with in the tongue.

DISEASES OF THE SALIVARY GLANDS.

Of the salivary glands, the parotid is by far the most frequently diseased. For this there does not appear to be any sufficient cause, but its exposed position renders it more liable to injury and to the effects of exposure to cold.

Mumps, or **epidemic parotitis**, is an infective inflammation of the parotid, with, in some cases, implication of the other salivary glands. It is most common in young patients, and is accompanied by considerable swelling and pain, with enlargement of the neighbouring lymphatic glands. Suppuration very rarely results, but metastatic orchitis is common, and in females the ovaries or breasts may become inflamed. These complications commonly arise towards the end of the attack. Occasionally, atrophy of one of the testes follows the orchitis.

Inflammation of the parotid occurs with considerable frequency as a complication of pyæmia, septicæmia, and other allied conditions; in such cases suppuration is common.

Salivary fistulæ.—A salivary fistula is a sinus in the cheek communicating with the duct of the parotid gland. It results from a wound implicating the duct, or from suppuration. The aperture of the sinus is usually very small, and may be so minute as to be difficult of detection, except by the escape of saliva. Such fistulæ are often prevented from healing by some obstruction in or around the duct. In other cases a salivary fistula may result from an extension of suppuration into some part of the gland itself. I have seen such fistulæ complicating suppuration in connection with necrosed bone, and in strumous disease of the lymphatic glands.

Salivary calculi are concretions of earthy material formed in the duct of one of the salivary glands, being most often found in that of the sub-maxillary gland

They are usually of a dirty-white colour, and have a rough surface. In shape they somewhat resemble a date-stone, being elongated and oval. They are usually very small, but may measure as much as an inch and a half in length. They consist chiefly of phosphate and carbonate of lime combined with a little animal matter; rarely, they are formed around a foreign body. As the result of the obstruction to the escape of saliva, the gland frequently becomes swollen during mastication, the swelling subsiding as the retained fluid obtains an exit. The tissues around the obstructed duct usually become indurated after a time, and the duct itself in some few cases has been found dilated into a cystic swelling behind the obstruction.

Tumours of the salivary glands are almost confined to the parotid, being rare in either the sub-maxillary or sub-lingual glands. The commonest tumour of the parotid is one composed of a mixture of cartilage, myxomatous tissue, and a varying quantity of connective-tissue cells and ill-formed glandular acini. Growths of this nature are often spoken of as "parotid glandular tumours" or "adenomata," but it is quite rare to find a growth the bulk of which is composed of true gland tissue. The cartilage met with in these tumours is of the hyaline variety, and it is by a mucoid degeneration of its stroma and of that of the fibrous tissue of the growth that the myxomatous tissue is produced.

In some cases there is a considerable admixture of sarcomatous tissue; in others, the growth is chiefly cartilaginous; and, as might be expected from this variety in structure, there is a corresponding difference in the rapidity with which such tumours develop. These growths show no marked preference for either sex, and are of frequent occurrence in adults of middle age; they are not so often seen in children. They grow most commonly in that part of the parotid which overlaps the lower jaw, are usually encapsuled, and, when small, are but little adherent to surrounding parts. If left alone, they may

attain a very large size, and may cause death by encroaching upon the pharynx or the large vessels in their neighbourhood. They do not affect the lymphatic glands, and, although prone to recur locally after removal, never become disseminated.

In addition to such tumours as these, which are common, the parotid is much more rarely affected by growths of a

FIG. 146.

Section of a Parotid Tumour. The specimen shows some ill-developed glandular tissue below, myxomatous tissue in the centre, and cartilage in the upper part. (Crouch, $\frac{1}{6}$ in.)

more malignant nature. Both sarcomata and carcinomata are met with in this gland, and, although they seldom become disseminated, they are very liable to cause death by pressure upon, or infiltration of, the various structures which lie in their immediate neighbourhood.

The only tumour which occurs in the **sub-maxillary** gland with sufficient frequency to deserve mention is one composed of cartilage. Even this is rare, not more than a few cases being on record. Both structurally and clinically they resemble enchondromata in other situations. They are therefore quite innocent.

CHAPTER LVII.

DISEASES OF THE TONGUE.

MACROGLOSSIA.

The enlargement of the tongue to which the name of macroglossia has been given is most frequently, but not always, of congenital origin, although in the majority of cases the organ continues its abnormal growth after birth. In this disease the whole tongue, or, rarely, a part of it, is enlarged, and in severe cases protrudes between the lips, or even overhangs the chin. On account of the constant pressure exercised upon the lower jaw, the latter is liable to become deformed, and the development of the teeth to be arrested. By reason of its exposure to injury and irritation, the swollen tongue is very liable to become inflamed, and each attack of glossitis leaves it larger than before. In extreme cases there may be great difficulty in breathing, whilst both speech and deglutition are necessarily much interfered with.

An examination of a tongue affected with this disease shows that the surface is dotted over with small semi-translucent vesicles or papules, which are much more numerous in some cases than in others. The organ is peculiarly shapeless, and may be scarred from old ulcerations, or else marked by the results of more recent inflammation. On section, the tissues are softer than natural, paler in colour, and with a marked absence of healthy muscle. Microscopical examination shows an increase of soft fibrous tissue, with the formation in parts of a true lymphatic

structure, such as is met with in the more cortical portions of a lymphatic gland, although of less regular formation. The whole organ is more or less infiltrated with leucocytes according to whether or not it has been the seat of recent inflammation, and its arteries and veins are frequently dilated and thickened. It will thus be seen that in macroglossia there is no true hypertrophy, but rather a general overgrowth and infiltration of the tongue with lymphatic tissue, lymph, and leucocytes, a condition which is generally complicated, and, indeed, to some extent promoted, by frequent attacks of inflammation. On account of this overgrowth of lymphatic tissue, Virchow has proposed the name "lymphangioma cavernosum."

GLOSSITIS.

The tongue is liable to be attacked by various forms of inflammation, some of purely local origin, others the result of some constitutional defects; some superficial and non-ulcerative; others ulcerative, and others, again, affecting the whole substance of the organ. The common causes of chronic glossitis are excessive smoking, drink, syphilis, and chronic dyspepsia.

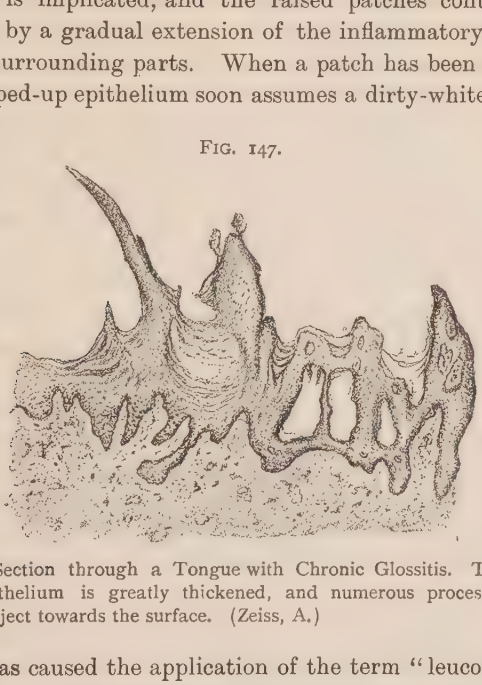
Superficial glossitis, unattended by ulceration, may be either subacute or chronic. It is very commonly of local origin, and may result from any form of irritation—*e.g.*, excessive smoking, scalding by hot liquids, chafing by rough teeth, &c. It is also frequently produced by gastritis or dyspepsia, and is common in connection with chronic alcoholism.

In the **subacute** forms, the surface of the tongue is very patchy. In parts it is covered by white fur; in other parts it is raw and glazed, as though the surface epithelium had been removed, and this is, in fact, what has really happened, for in glossitis there is a catarrh, with desquamation of the epithelial covering. The papillæ appear larger than natural, and the organ is painful and tender.

In the more **chronic** forms of glossitis, there is at first

a reddening of the inflamed area, which is hidden to a great extent by a thick covering of fur; this is gradually followed by thickening of the surface epithelium and the formation of raised white patches of varying size and shape. In some cases the whole or the greater part of the surface of the tongue is implicated, and the raised patches continue to enlarge by a gradual extension of the inflammatory process to the surrounding parts. When a patch has been formed, the heaped-up epithelium soon assumes a dirty-white colour,

FIG. 147.



Section through a Tongue with Chronic Glossitis. The epithelium is greatly thickened, and numerous processes project towards the surface. (Zeiss, A.)

which has caused the application of the term "leucoplakia," whilst, on account of the thickening, the disease has been named "ichthyosis" or "psoriasis linguæ." In many cases the thickening subsides on the removal of the cause, but in other cases it continues, and, the epithelium becoming dry and horny, the surface is soon cracked and fissured. Microscopical examination shows more or less cell infiltration of an inflammatory nature, with great increase of the corneous layer. In some cases the interpapillary processes of epithelium are much increased in size; in others, they are themselves broken up by cell infiltration. This condition,

if allowed to progress unchecked, is liable occasionally to terminate in epithelioma.

The smooth, glazed tongue, which affords another instance of the results of inflammation, is also the product of a chronic superficial glossitis. There is often no apparent cause for the disease, either constitutional or local, and the affection is peculiarly persistent, and obstinate in its resistance to treatment of all kinds. In such cases the surface of the tongue is bright red, perfectly smooth, and shiny, presenting no papillæ, and looking very much as though it had been brushed over with some transparent varnish. This condition usually gives rise to a burning and smarting sensation, and is occasionally accompanied by increased salivation. It affects women as well as men.

In connection with the subject of superficial glossitis, the **rashes** which are met with on the tongue may be briefly mentioned. The commonest of these is a simple white discoloration, which often extends to the gums and mucous surface of the lips; it appears to be commonly caused by smoking, and is very transient. Much less common than this is the **annulus migrans**, or **wandering rash**, which is characterised by the appearance of light-coloured crescentic or circular bands or patches, spreading eccentrically, rapidly fading in one place, and equally rapidly appearing in another, very prone to persist for months or years in spite of treatment, causing no symptoms of importance, and owning no definite cause. It may occur at any age, and affects both males and females.

Acute parenchymatous glossitis.—This is, fortunately, a rare affection. It may follow injury, such as the biting of the tongue in an epileptic fit, scalds and burns, stings by insects, &c., or may occur during mercurialism, or in the course of one of the specific fevers. In other cases it is attributed to exposure to cold, or to wounds by dirty and septic instruments.

The disease is characterised by a general swelling of the whole organ. This occurs with much rapidity, and may

cause so great an enlargement that the tongue protrudes from the mouth. The surface is livid and shiny except where it is exposed beyond the lips, whilst in advanced cases the lividity may increase till the colour deepens to a blue-black. Suppuration is rare, but in some cases superficial sloughing has occurred. If untreated, the disease may prove fatal by causing dyspnœa, for, not only is the whole tongue swollen, but the ary-epiglottic folds also soon become œdematous. When the inflammation once commences to subside, the organ rapidly diminishes in size.

Abscess of the tongue is rare. It is probably invariably of local origin, and never attains any considerable size. I have seen symmetrical abscesses form in the tongue after an attack of facial erysipelas which extended to the buccal cavity. These abscesses developed without much pain, and there was at no time any material swelling of the whole organ such as is found in typical acute glossitis.

The **Syphilitic** affections of the tongue have already been described in the chapter on Syphilis, on page 146.

ULCERATION OF THE TONGUE.

Ulceration of the tongue occurs under several forms, and owns various causes. **Simple** single ulcers due to irritation of sharp teeth or of rough pipes are most common on the lateral margins. The shape is irregular; the base sometimes sloughy, at other times covered by pus; the edges sharply cut, except when healing is in progress; and the tongue around not indurated. The ulcer is often very painful and tender, and is accompanied by excessive salivation.

Follicular ulcers of the tongue, due to dyspepsia and other allied conditions, are frequently multiple. They are generally not limited to the tongue, but are found also upon the floor of the mouth, the palate, and the lips. The ulcers are superficial, circular, about the size of a split pea or less, and readily heal on the removal of the cause. This form

of ulcer is relatively common in young children suffering from teething.

Ulceration of the tongue of a foul and sloughy nature, and associated with similar conditions of the contiguous mucous surfaces, occurs also in connection with mercurial poisoning and with scurvy.

Tubercular ulceration of the tongue has only of late years been brought into notice. Like other tubercular lesions, it is most common in young adults, though cases have been recorded at forty years and upwards. Most commonly there is evidence of tuberculosis of the lungs, either in the active stage or else temporarily quiescent. The tubercular ulcer commences as a little nodule, which soon becames raw on the surface, and slowly ulcerates; in some cases no nodular thickening precedes the sore. These ulcers are most common at the tip of the tongue or on the dorsum in the middle line. The shape is irregular, the surface sloughy, and the discharge thin and watery; the edges are ragged and undermined, and there is an almost complete absence of induration. When occurring near the tip, there is sometimes an appearance as though the extremity of the tongue had simply been rubbed away and a raw surface produced. Sometimes, under treatment, these ulcers show a tendency to heal, but more commonly this is not the case, and the sore remains open and indolent, neither increasing nor diminishing to a noticeable extent for weeks or months. More rarely the ulcer rapidly extends, and in one severe case, whose progress I watched, the whole of the dorsum of the tongue, the soft palate, the tonsils, and the ary-epiglottic folds were successively involved. The lymphatic glands are not usually affected, but in some cases they become enlarged and inflamed. The prognosis of such cases is bad, chiefly, perhaps, on account of the usual occurrence of tubercle in other parts; but some cases in which early excision has been practised have done well.

In connection with tubercle of the tongue, the occasional

occurrence of lupus may be mentioned. It is extremely rare, and requires but brief notice. In the cases I have seen the ulceration was associated with similar disease of the nose and face.

TUMOURS OF THE TONGUE.

The most common tumour of the tongue is **epithelioma**, and for this reason it is here given the first place. Epithelioma is much more frequently seen in men than in women, and usually occurs after forty years of age. It is often preceded by some form of chronic inflammation, such as simple ulcer due to bad teeth, syphilitic ulcers, chronic superficial glossitis, &c. Considering the frequency with which it follows syphilitic lesions, the greatest care should always be exercised in examining a tongue which has long been the seat of syphilitic disease, for it is quite possible that, although the ulceration is syphilitic, it is also something more, and that epithelioma has been engrafted upon the pre-existing ulceration. Occurring in a tongue not previously ulcerated or inflamed, epithelioma usually commences as a thickening of the surface epithelium in the form of a warty growth, pimple, or nodule. As this nodule increases in size, its most central and superficial part breaks down and forms an ulcer, which is sometimes of very characteristic appearance. In other cases the disease commences as a crack or fissure, which slowly increases in depth, whilst its edges become thick and everted. By far the most common place for an epithelioma is the lateral margin of the tongue, and after that the under surface; but, when attacking the site of a pre-existing ulceration, it often commences on the dorsum. The epithelial ulcer is of irregular shape, with a sloughy, grey base, and everted edges which are raised above the surrounding surface and are very hard. If the subjacent tongue be felt between the finger and thumb, it will be found to be much indurated; and if a section of it be made, it will be seen that this induration is due to the infiltration of the muscle by the

new growth. The ulcer, in fact, is an ulcer of a new growth situated on and in the tongue, and it is bounded on all sides, not by lingual tissue, but by the growth itself. It is the presence of this tumour, often raised considerably above the surface, with edges overlapping and mushroom-like, that especially characterises the epithelial ulcer, and when we speak of the indurated edges and base we really are talking of the feel of the tumour itself. If allowed to run its course unchecked, the growth continues to infiltrate the subjacent structures. It gradually extends to the floor

FIG. 148.

A Tongue with a large Epithelioma on its Left Lateral Margin. The centre of the growth has ulcerated.

of the mouth, and so fixes the tongue that all movement is rendered difficult. It involves the soft palate and the alveolar margins, the maxillary bones, and, passing backwards, it infiltrates the epiglottis or even the larynx itself. As the growth extends, so also does the ulceration; and in some cases the latter progresses quite as rapidly as does the tumour, so that just as fast as the latter grows, just as quickly does it disintegrate on its surface. In some cases there is much hæmorrhage, and in all there is a bloody discharge, which is often very foul. Salivation is profuse, and pain is constant and severe.

The sub-maxillary **lymphatic glands** are early implicated. At first they are hard and movable; soon they become fixed, and soften in their centres. If examined after removal, they are found to be infiltrated with epithe-

lial new growth, which soon extends from them to the surrounding tissues, so that, in advanced cases, large masses are formed in the neck extending all round the lower jaw, fixing it so as to greatly limit its movements, and passing down the neck even as far as the clavicle.

The inevitable termination of such cases is a most painful and lingering death, which is due to the exhaustion induced by constant pain and sleeplessness, with inability to eat, and in some cases is accelerated by loss of blood from the ulcerated surface. Very frequently broncho-pneumonia is set up by the passage of the inspired air over the foul surface in the mouth, and in some instances the lungs may be found almost gangrenous. Dissemination is not very common, but I have seen numerous secondary growths in both lungs and in the liver.

If an epithelioma of the tongue be excised, or even if the whole organ be removed, recurrence is unfortunately frequent. It by no means follows that a patient is never permanently freed from the disease, for cases occur in which many years pass by without recurrence, and the patient dies of some other and separate affection. But, speaking of epitheliomata as a class, it must be confessed that those of the tongue are more malignant than similar growths occurring in most other parts. Thus, they grow much more rapidly, ulcerate more quickly, and affect the lymphatic glands much sooner than do epitheliomata of the lip, and are, in addition, more prone to recur and to disseminate. It may be that this clinical history depends in part on the favourable conditions for growth, namely, the constant, even temperature and moisture of the mouth; but these are probably only of secondary importance, for it must always be remembered that tumours growing under precisely similar conditions vary much in their individual malignancy according to the tissue or organ in which they originate.

Malignant tumours of the tongue other than epitheliomata are very rare, and need only to be mentioned. The other

varieties of carcinoma are practically unknown, and but a few cases of sarcoma are on record.

Innocent tumours of the tongue are also comparatively uncommon, and perhaps that one which is most often seen is **nævus**. This, like similar growths elsewhere, is usually noticed soon after birth, and may either continue to grow, or, after remaining stationary for a time, may slowly wither and disappear. On account of the thinness of their covering, and their liability to injury by hard substances during the act of mastication, nævi of the tongue are rather liable to bleed, and occasionally do so to a dangerous extent. In rare instances they attain such a size as to seriously interfere with deglutition and speech.

Columnar-celled adenomata occur as rare tumours situated deeply in the substance of the tongue, in front of the epiglottis, and covered by normal epithelium. They probably originate either in the glands met with in this situation, or else in the remains of the lingual duct. They consist of tubes and acini lined with columnar epithelium, and occasionally contain cysts which enclose a clear sticky fluid. The tumours occur in young adults and appear to be innocent. I have seen two cases in women between twenty and thirty.

Cysts of the tongue containing a mucoid fluid, and due to the distension of a mucous follicle, are occasionally seen. They are seldom larger than a pea, but may attain a greater size.

Papillomata of the tongue are found on all parts of its dorsum. They may be seen at all ages, and are sometimes congenital. They occur as small raised growths, tending to be pedunculated, with roughened surfaces covered by thickened epithelium, not ulcerated, and not infiltrating the substance of the organ. They consist of an overgrowth of the connective tissue which underlies the epithelium, and are covered by an abnormally thick layer of the latter. Other papilloma are also found which are of lymphatic origin and are properly classified as **Lymph-**

angiomata. In these a part of the tongue presents a number of small raised papules or vesicles which contain clear fluid. These growths are often, but wrongly, called degenerate nævi. They are really localised overgrowths of the lymphatic tissue such as is also met with in macroglossia (see p. 515).

Amongst other and still rarer growths, **fatty** tumours and soft **fibromata** may be mentioned. They usually occur in the substance of the tongue itself, but cases are on record in which the fibrous growths have been pedunculated, and I have myself removed such a tumour from a child. Enchondromata and hydatid cysts have also been found in the tongue.

CHAPTER LVIII.

DISEASES OF THE NOSE.

Lipoma nasi.—With the exception of lupus and rodent ulcer, which have been already described, the only noteworthy disease peculiar to the cutaneous surface of the nose is the so-called "lipoma nasi." The growth to which this name has been applied is not, however, composed of fat. It results from acne rosacea, and consists chiefly of greatly enlarged sebaceous follicles, with thickening of the true skin by fibrous tissue of inflammatory origin. The growth is most common near the tip of the nose. It is usually lobulated and firm, and in some cases the lobules are pedunculated. Growths of this nature are most common in people of intemperate habits.

Deviation of the septum nasi, and partial displacement of the nasal cartilages, are generally of traumatic origin, but in many cases tend to increase. They are also in some cases of congenital origin, or develop about the period of puberty, and in such cases are frequently accompanied by bony or cartilaginous outgrowths from the septum. Occasionally, the bulging of the deviated septum causes so great a prominence in one nostril that it may be mistaken for a polypoid growth.

Simple catarrhal inflammation of the mucous lining of the nostrils is quite the commonest of the affections to which these passages are liable. The most common cause of acute catarrh or coryza is exposure to cold, and the changes seen in the mucous membrane are such as are typical of similar affections of other mucous surfaces.

Chronic coryza is in some cases dependent upon the

presence of foreign bodies introduced from without, or of nasal calculi or tumours. In other instances it is dependent on "hay-fever," and more rarely on gout. The purulent variety is most common in children, and often results in much permanent thickening of the mucous membrane, with partial obstruction to respiration. In the cases dependent on syphilis the discharge is usually profuse and purulent, often foul-smelling, and accompanied by ulceration; but all cases are not so severe.

"**Hypertrophic rhinitis**."—This term is applied to enlargement of the turbinate bodies, and most often affects the inferior one. It usually results from some interference with nasal respiration, and thus is often seen as a complication of adenoid growth and of deviation of the nasal septum. Beyond causing stoppage of the affected nostril and running from the nose, it gives rise to no definite symptoms and causes no secondary changes in the nostrils.

Atrophic rhinitis.—This disease commences in childhood as a simple purulent catarrh, and it is probable that when properly treated at this stage it can be completely cured. If untreated, however, the inflammatory process results in a gradual destruction of the normal mucous membrane and submucous tissue, and in atrophy of the turbinate bodies. In this stage the chief symptom is ozæna of the worst type, and an examination of the nostrils reveals the presence of dread scabs and crusts and enlargement of the nostrils, the destructive process affecting in time the turbinate bones themselves. A condition such as this is incurable, and often leads to pharyngeal catarrh and chronic laryngitis.

Ozæna.—The term ozæna is used to designate a condition in which there is a discharge of pus from the nostrils accompanied by a peculiarly foul odour. The discharge is often bloody, and may be mucoid rather than purulent; in all cases it is fetid.

Ozæna results most often from atrophic rhinitis, but also from various forms of ulceration, and may be of traumatic,

scrofulous, or syphilitic origin. Scrofulous ulceration is most common in children, and especially in those who exhibit some other signs of struma. The ulcers are small, but multiple, are usually covered by a scab, and secrete the thin watery pus characteristic of scrofulous inflammations in other parts. In some cases the ulcers are of a lupoid nature. Syphilitic ulcers are sometimes seen in the subjects of congenital syphilis, but are usually met with in adults; they also are multiple, and may be either superficial or deep. The ulcers are larger and less numerous than those of scrofulous origin.

In both syphilitic and scrofulous ulceration the subjacent bone may be diseased, and either caries or necrosis may complicate the case. In some instances there is extensive destruction of the osseous framework—a condition which is most common in syphilitic patients. In cases of ozæna following injury, it will generally be found that there is some necrosis, and it is to the presence of the dead bone that the continuance of the discharge is to be attributed. In other cases the ozæna is dependent upon the introduction and retention of foreign bodies, or on the presence of nasal calculi. These latter bodies, called also **rhinolithes**, are most commonly found in the inferior meatus, and are often formed around a foreign body. They consist chiefly of phosphates and carbonates of lime and magnesia.

TUMOURS OF THE NOSTRILS.

The commonest tumours of the nostrils are **mucous polypi**. These growths occur for the most part in young adults, and in some cases follow chronic coryza. They are almost invariably multiple and pedunculated, and vary in size from that of a pea to that of a raisin. In colour they are pink or red, and have a translucent appearance. Their surface is very smooth and slippery, and their consistence soft and elastic. Microscopically examined, they are found to be composed of myxomatous tissue, or of very soft and succulent fibrous tissue, in the meshes of which there is a

mucoid fluid; they are covered by ciliated epithelium such as is normally met with in the nares. After long exposure near the nasal orifices, the most dependent parts of these growths become more firm, fibrous, and white. The usual situation for these growths is the external wall of the nostril, where they are commonly attached to the middle turbinate bone, but they are also found on the superior and inferior turbinate bones, as well as on the mucous membrane covering the roof of the nose; they are very rarely attached to the septum nasi.

The irritation caused by the presence of these growths usually sets up a nasal catarrh, which, in its turn, probably promotes the growth of polypi, for there can be no doubt that the longer polypi are left untreated the more numerous do they become. The smaller and more recent ones occupy the upper parts of the nasal cavities, and do not develop or come into view until the older and more superficial ones have been removed. In all cases polypi cause an unpleasant nasal intonation of the voice, and, when numerous and large, may cause bulging and deformity of the nose. However large they grow, or however long they remain, they scarcely ever bleed spontaneously.

Fibrous and sarcomatous polypi.—These growths are not so common as are the mucous polypi; they tend to grow especially from the roof and back of the nose and the septum. They spring also from the roof of the pharynx, being attached to the periosteum of the base of the skull. They occur at all ages, but are, perhaps, more common in the young. They usually originate in the periosteum, and not in the mucous membrane, as do the simple myxomata. They vary in size, but often grow so large that the nostril is no longer able to contain them, and they protrude into the pharynx; when occupying this position, they are known as naso-pharyngeal polypi. Their surface is in some cases smooth, but in others papillated or spongy. On microscopical examination, they are found to consist either of simple fibrous tissue or else of a fibrous matrix containing connective-

tissue cells of various shapes. In some cases the whole growth is sarcomatous, and composed entirely of cells. In the more fibrous and slowly-growing tumours, which occur especially in boys or young adult males, there is often a very large development of blood-vessels and vascular spaces, constituting a true angiomatous growth. Occasionally this tissue is so much developed as to appear almost like an arterio-venous aneurysm.

Growths such as these grow with very varying degrees of rapidity, but those composed of pure fibrous tissue increase more slowly than the pure sarcomata. As they develop, they tend to extend into neighbouring cavities, and sometimes infiltrate the surrounding tissues, as do malignant growths elsewhere. They may thus produce considerable deformity. Hæmorrhage is a common symptom, and may be very excessive. If removed, all growths of this class show some tendency to recur, and, although this is most marked in the tumours of sarcomatous structure, it is seen also in those composed of simple fibrous tissue.

Columnar-celled epithelioma occasionally develops in the nostril, usually attacking the roof and growing slowly; it is very little liable to affect the glands, but commonly causes a good deal of hæmorrhage.

Tumours of the nostrils other than those above described are of rare occurrence, but papillomata, osteomata, and enchondromata growing from the nasal cartilages have occasionally been observed.

ADENOID VEGETATIONS.

Adenoid vegetations are overgrowths of the adenoid or lymphatic tissue of the naso-pharynx, and are often associated with a general thickening of the mucous membrane. They occur as small, pendulous growths, seldom larger than a pea, and are generally sessile, though occasionally pedunculated. The mucous membrane of the pharynx, of the soft palate, and of the posterior nares is the seat of the disease, and in many cases the growths extend around the

orifices of the Eustachian tubes. A microscopical examination shows that the vegetations are covered by mucous membrane, and are composed of a framework of loose connective tissue, well supplied with vessels, in which are embedded lymphatic follicles such as are found in the tonsils.

Adenoid vegetations are essentially a disease of childoood, and occur with about equal frequency in the two sexes. They are considered by some authors to be relatively common in strumous subjects, and are often seen in conjunction with enlarged tonsils. The chief trouble occasioned by these growths is deafness, which results partly from nasal obstruction and the mechanical occlusion of the Eustachian orifices, and partly from an extension of congestion and catarrhal inflammation along the Eustachian tube to the middle ear. Other troubles are an unpleasant nasal voice, attacks of pharyngitis, laryngitis, and nasal catarrh. It is probable that, as the patients grow up, the vegetations shrink and atrophy, but in many cases, before this, which may be considered the natural cure has occurred, permanent deafness has resulted.

CHAPTER LIX.

DISEASES OF THE ŒSOPHAGUS.

The œsophagus is very rarely the seat of simple inflammation or ulceration. Either of these conditions may, however, be set up by injury caused by swallowing rough or sharp substances, or else by drinking caustic acids or alkalies. In diphtheria, also, the ulceration and formation of membrane frequently extend to the œsophagus.

Slight inflammations or ulcerations clear up without inducing any important change, but in some cases of poisoning by corrosive liquids the scarring which ensues is sufficient to cause a diminution in the calibre of the tube, and so produce a **fibrous stricture.**

These, like strictures of other mucous passages, are very liable to continue to contract, and sometimes cause such narrowing that the passage of food becomes impossible, and death from starvation ensues. In cases such as these, the œsophagus above the strictured portion is usually dilated and its walls often thickened. This condition readily explains the fact that food is sometimes apparently swallowed, and is after a time regurgitated, having merely gone so far as the pouch above the stricture. In other cases, again, the mucous lining of the pouched portion is inflamed or ulcerated, with the resulting discharge of much mucus, which is constantly being "hawked up" by the patient.

Cancerous stricture is unfortunately much more frequent than simple fibrous narrowing, and is especially

liable to occur in old people. It is more often met with in men than in women. The most common variety of malignant growth of the œsophagus is certainly epithelioma, but scirrhous carcinoma is by no means rare, and I have myself seen four or five instances of it. Colloid and medullary cancers are much more uncommon.

Epithelioma is especially frequent in the upper part of

FIG. 149.

Dilatation of the œsophagus above a stricture.

the œsophagus, and is met with just behind the cricoid cartilage more often than in any other situation. In some cases the new growth extends into the lumen of the tube, and thus obstructs the passage of food, but more frequently it infiltrates the walls in their whole circumference, and, gradually causing an induration and shrinking of the latter, results in the formation of an "annular stricture." In other instances, again, the growth extends along several

inches of the tube, and so causes a "tubular stricture." In any case the mucous membrane soon becomes ulcerated, and a foul, sloughing, and bleeding surface is produced. The other most common sites for epithelioma are the lower portion of the œsophagus just above the cardiac orifice of the stomach, and that part of the tube opposite to the bifurcation of the trachea.

Scirrhous carcinoma is not so common in the upper part of the œsophagus as lower down, and is more often seen in that part of the tube which is within the thorax than that which is in the neck. The growth presents the characteristic appearance of similar growths elsewhere, being extremely hard, causing much contraction, and developing with comparative slowness.

Both epithelioma and scirrhus of the œsophagus terminate fatally, and each in much the same manner. Death may be finally brought about in one of several ways. In some cases the inability to swallow induces starvation, but in many more the growth extends to, and opens, the trachea or bronchi, sets up septic broncho-pneumonia by the passage of the foul discharge into the air-passages, and sometimes leads to an almost gangrenous condition of the lung. More rarely the pericardium is involved, and acute pericarditis terminates the case. In other cases, again, death appears to result rather from blood-poisoning than anything else, and the source of this is not difficult to find when we consider the extremely foul surface from which discharge is constantly passing into the food-passages or is being directly absorbed by the lymphatics.

If a patient who has died of cancer of the œsophagus be examined post-mortem, it is by no means infrequent to find no glandular enlargement and no secondary growths; but in other cases, the glands in the neck or the thorax are infiltrated, and in some the liver, kidneys, or lungs are similarly affected.

Sarcomatous tumours of the œsophagus are very rare, and do not require any special description.

CHAPTER LX.

HERNIA.

A HERNIA is an abnormal protrusion of some of the contents of the abdominal cavity. This protrusion most often occurs at one of the places where the passage of various structures from the abdomen to the lower extremities or genital regions provides apertures which may allow, in addition, the transit of some of the abdominal viscera.

A hernia is described as being composed of a **sac** and **contents.** The **sac** consists of peritoneum, and, except in the case of congenital malformations, is formed by a protrusion of a portion of the parietal peritoneum in front of the viscus which is escaping from the abdomen. At first this sac has no independent existence—*i.e.*, if the contents are returned into the peritoneal cavity, the bulging peritoneum will again become smoothed out. This, however, is only in the very earliest stages of the hernia, for after a short time the sac contracts adhesions to the tissues amongst which it has been thrust, and can no longer be reduced. And not only does the sac become adherent to the tissues around it, but the folds into which the peritoneum is thrown at the orifice through which it is extruded become adherent to one another; for it is evident that, if the peritoneum covering the internal abdominal ring, for example, be thrust down into the scrotum, it will be thrown into numerous folds where it passes through the ring, and it is between these folds that adhesions shortly form. This narrowed part of the sac which lies close to the abdominal

cavity is named the **neck,** whilst the lower part, which is much more capacious, is called the **fundus**. The sac thus becomes somewhat flask-shape, and the neck is in time still further narrowed. For, as the result of the pressure of its contents within, and of the abdominal rings without, the peritoneum and sub-peritoneal tissues become matted and thickened by fibrous tissue, and a more or less dense fibrous ring, with occasionally some unstriped muscle, is formed at

FIG. 150.

The Sac of an Inguinal Hernia, showing the constriction and fibrous ring at the neck.

the neck. The peritoneum forming the fundus also undergoes a change of texture. In most herniæ (*e.g.*, inguinal and femoral) it becomes slightly thickened; whilst in others, notably in the umbilical variety, the peritoneum forming the sac is so thinned that in places it entirely disappears, and the sac is then called "incomplete." In other herniæ, again, the sac is incomplete from the first, for a viscus which is not entirely covered by peritoneum, such as the bladder, or in some cases the cæcum, may escape from the abdominal cavity without pushing a complete covering of peritoneum in front of it. In such herniæ the protruded viscera rapidly become adherent to the tissues amongst which they lie, and are consequently irreducible.

The **contents** of a hernial sac consist of a little fluid

secreted by the serous surface, with, usually, either intestine or omentum; but almost any of the abdominal viscera may herniate, those only being excepted which, like the pancreas, are firmly fixed. A hernia containing omentum is named an **epiplocele**; one containing intestine, an **enterocele**.

The **condition of the contents** varies. When the latter can be returned within the peritoneal cavity, the hernia is said to be **reducible**, whilst if such is not the case it is called **irreducible**. In some cases, usually as the result of injury, the hernia becomes **inflamed**; and in others, fæces, accumulating in the gut which is in the sac, cause the hernia to increase in size and to become for the time irreducible—a condition to which the term **obstructed** or **incarcerated** is applied.

Lastly, the hernia may be **strangulated**, but before describing this it is necessary to say a few words on the subject of irreducible herniæ.

As already mentioned, some herniæ are irreducible from the time of their formation, a circumstance which is dependent on the incompleteness of their sac; whilst others, which are at first reducible, become irreducible through the thinning away of the peritoneum forming the sac, and the subsequent adhesion of the contents to the tissues with which they are thus brought into contact. The commonest cause of irreducibility, however, is thickening of the protruded omentum, for, on account of the friction and pressure to which a herniated piece of omentum is subjected, it soon becomes gradually indurated and increased in size by the formation in it of fibrous tissue, and is consequently after a time unable to return by the aperture through which it formerly escaped. In other cases, again, this thickening is accompanied by the contraction of adhesions to the sac-wall or to some of the contained intestine, and thus a further obstacle is offered to reduction. Lastly, it has been already mentioned that, when the intestine in a hernial sac becomes blocked by fæces, it is for the time irreducible.

In a **strangulated** hernia, not only are the contents irreducible, but they are so tightly constricted that the *circulation of the blood through them is interfered with ;* it is to the latter condition that by far the greater importance is to be attached. In some cases, and especially those where the hernia is suddenly caused by exertion or injury, the contents, as soon as extruded, are so tightly gripped by the margins of the aperture through which they are thrust that strangulation at once ensues. This is by no means a

FIG. 151.

A Hernial Sac, containing a mass of thickened omentum which has become adherent to the posterior wall of the sac.

common occurrence, and it is usually only after existing for some time that herniæ become strangulated. At first sight this is not altogether easy of explanation, for it does not appear evident why a hernia which has frequently descended before, and has always hitherto been reducible, should suddenly become so tightly gripped as to cause its irreducibility and constriction. And it may be easily seen that these conditions cannot be due to any sudden alteration in the size of the rings or the neck of the sac, for neither of these possesses the requisite amount of contractility. The true cause of the strangulation is probably to be found in

the descent of a greater quantity of intestine or omentum than has before descended, and the consequently greater pressure to which either is subjected at the aperture through which it passes. This is borne out by the evidence of patients, who often state that on the occasion of strangulation the hernia has descended in greater bulk than previously. The descent may be due either to exertion or to excessive movement of the intestines, as in colic or diarrhœa.

The position of the constriction relative to the contents of the sac differs in different cases. In some it is outside the sac—*e.g.*, at the external or internal abdominal rings, at Hey's or Gimbernat's ligaments, &c. In others it is the fibrous thickening already described as forming the neck of the sac which is the constricting element; whilst in a few and rare instances the coils of intestine are encircled by bands of adhesion formed within the sac itself, in the manner already mentioned in connection with irreducible herniæ.

If intestine be strangulated, it is the passage of venous blood which is first interfered with, and the gut becomes congested. The congestion in its turn causes swelling, and thus increases the tightness of the constriction. The gut becomes of a dark plum colour, very tense and shiny, and at the same time the fluid in the sac quickly increases in quantity and becomes blood-stained, being exuded from the serous surface of the imprisoned intestine. In many cases the distended vessels relieve themselves by rupturing into the cavity of the gut, the blood being subsequently passed *per anum* if the strangulation be relieved. After a time, not only is the venous circulation obstructed, but, as the tightness of the constriction increases, the flow of blood through the arteries is arrested, and gangrene of the imprisoned intestine shortly ensues. In such a case, the colour of the gut changes to an ashen grey, the surface becomes wrinkled, its shiny appearance is lost and the peritoneum can be peeled off; finally, rupture results, and

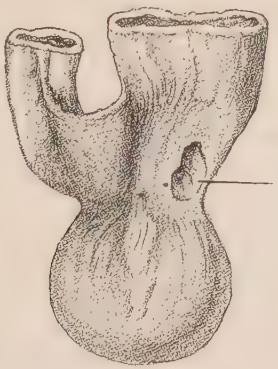
fæces escape into the sac. Usually, the patient dies by the time such a stage has been reached, death being usually brought about by a combination of circumstances, chief of which is the exhaustion which results from the enforced starvation and the pain; but the absence of proper abdominal respiration, and the pushing up of the diaphragm by the distended intestines in many cases causes very serious congestion of the lungs and distension of the right side of the heart. In very rare instances the patient survives yet longer, and, if such be the case, suppuration and sloughing of the sac and of the tissues around it will follow, and fæces will finally be discharged through the skin. In some cases peritonitis supervenes, being caused by an extension of inflammation from the sac; but in other cases, even when gangrene ensues, the extravasation of fæces into the peritoneal cavity, and the spread of inflammation to the serous membrane are arrested by adhesion between the strangulated gut and the neck of the sac.

But not only does the constriction of the gut tend to cause sloughing of the part beyond the stricture; it also causes ulceration of the intestine at the seat of stricture itself. This is a fact of the greatest practical importance, for it may happen that the state of the knuckle of intestine which is found in the sac during the operation of herniotomy is sufficiently good and free from appearance of sloughing to warrant its return after division of the stricture, but that, nevertheless, the intestine *where gripped* is at one small spot already ulcerated or sloughing. If this condition is not ascertained by drawing down the gut and examining it before effecting its reduction, nothing can prevent subsequent fæcal extravasation and diffuse peritonitis. (See Fig. 152).

In cases where an opportunity is afforded of making a post-mortem examination of a case of strangulated hernia which has not been relieved by operation, the difference in the appearance of the intestine above and below the seat of strangulation is very marked, for the coils above are

greatly distended and congested, and in extreme cases, where the distension has been excessive, the peritoneal coat splits; the coils below the stricture, are, on the contrary, collapsed and pale.

FIG. 152.



Loop of Intestine from a case of Strangulated Femoral Hernia. A small oval ulcer marks the place where the gut has been constricted by Gimbernat's ligament. The large piece of intestine to the right is a portion of the dilated intestine above the seat of strangulation; the small piece of intestine to the left was below the stricture.

In those cases where the gut has sloughed and all the fæces continue to be discharged through the skin the patient is said to have an **artificial anus.** When most of the fæces are passed by the anus and but little fæcal matter escapes by the opening, the term **fæcal fistula** is employed. If only a small portion of the whole circumference of the gut has been destroyed, such an opening may gradually contract and finally close, but when the sloughing has been of greater extent so fortunate a conclusion is not to be expected. In consequence of the pressure exercised by the abdominal contents on the distal portions of the intestine, the latter remains collapsed and does not permit of the passage of fæces, whilst, after a time, a further obstacle is interposed in the form of a protrusion of

a fold or spur of mucous membrane from between the apertures of the upper and lower pieces of intestine. This acts as a valve, and effectually prevents the passage of fæces by the natural channel, and, until it has been removed by operation, the artificial anus remains patent.

The causes of herniæ are either congenital or acquired. Of the former, the chief predisposing cause is a failure of the normal closure of the processes of peritoneum which in fœtal life extend into the scrotum and through the umbilical aperture. The consequent weakness of the abdominal wall is in some instances supplemented by an undue length of the mesentery, which allows the intestines to hang more heavily than is natural against the parietes. In a considerable percentage of such cases, the hernial tendency appears to be hereditary.

In later life the mesentery and omentum may acquire an undue length through stretching, consequent upon the accumulation of fat in their substance, and, from the same cause, the tension of the abdominal contents may be considerably increased; in some cases also the whole mesentery is displaced and slid downwards by the increased weight it is called on to support. It is thus that umbilical herniæ, more especially, are found in people over middle age who have recently become obese. The reverse condition also, namely, wasting and loss of flesh, tends to the development of herniæ, although in a different manner. Here, the abdominal contents become prone to extrusion on account of the removal of the fat and cellular tissue which normally occupy the apertures of exit from the abdomen. It will therefore be seen that both emaciation and obesity are causes of herniæ in adult life, and it may be added that anything which induces a loss of muscular tone, any condition of debility, is favourable to the production of a hernia. For, normally, the muscular walls of the abdomen by their constant contraction both support the contained viscera and promote the closure of all apertures which naturally exist.

As directly exciting causes of herniæ may be mentioned the constant straining cough of chronic bronchitis and wounds and lacerations of the abdominal walls. Thus, it is common for a hernial protrusion to follow the operations of ligature of the iliac vessels, abdominal section, &c., as well as accidental injuries of various kinds. Suppuration also tends to produce the same result.

VARIETIES OF HERNIÆ.

It is not possible to devote sufficient space in the present work to a description of the anatomy of the parts concerned in the various forms of herniæ; for such details, works on anatomy should be consulted. It is, however, desirable to point out very briefly the common positions of hernial protrusions, as well as the main points worthy of notice in connection with the probable seat of strangulation and the mode of formation of the sac.

In **inguinal** hernia the protrusion passes either through the whole length of the inguinal canal (an oblique hernia) or directly through the external abdominal ring (a direct hernia) and thence into the scrotum or labium. The sac of such a hernia may be (*a*) congenital or (*b*) acquired. Of the former there are several varieties.

Normally the descent of the testis into the scrotum is followed by a closure of the process of peritoneum which the organ carries with it from the abdominal wall. This closure commences, above, at about the level of the external abdominal ring, and, below, immediately above the testis. The intervening or "funicular" portion of the tunica vaginalis is subsequently gradually obliterated. If this closure does not occur at all, then the cavity of the tunica vaginalis testis communicates directly with the peritoneal cavity; and if a hernia descends, which is by no means necessarily the case, it will pass into a sac already formed, namely, the tunica vaginalis testis. Such a hernia is named "**congenital.**"

In other cases the tunica vaginalis testis becomes shut

off from the general peritoneal cavity by adhesion immediately above the testis, as already mentioned, but instead of the funicular portion of the peritoneal sac being obliterated, and closure occurring at the external ring, the funicular portion remains open and ready to receive any viscus that subsequently protrudes. To such a hernia the name of "**funicular**" is applied, for the sac is formed of

FIG. 153.

Diagram of a Congenital Inguinal Hernia. The loop of gut is within the tunica vaginalis testis.

FIG. 154.

Diagram of a Funicular Hernia. The loop of gut is in the unobliterated funicular portion of the tunica vaginalis, which has been separated from the testicular portion.

the unobliterated "funicular" portion of the tunica vaginalis, the testis itself being separated from the hernial sac by a thin septum.

In yet other cases both the funicular and testicular portions of the tunica vaginalis remain unobliterated and continuous with one another, but their cavity is separated from the general peritoneal cavity by the closure already mentioned, which normally occurs at the external abdominal ring. If a hernia now develop, the hernial sac is usually formed by a protrusion of a fresh portion of peritoneum from the neighbourhood of the internal abdominal ring, the sac being thus an acquired one. In such a case the hernial sac is liable to be protruded behind the un-

obliterated funicular portion of the tunica vaginalis, and as it increases in size, tends to bulge into the cavity of the latter. A hernia with such a relation to the tunica vaginalis is named "**infantile**."

Another variety of hernia is generally described which is said to be developed in connection with a precisely similar abnormal condition of the tunica vaginalis to that last described. It is named "**encysted**," and the sac is

FIG. 155.

Diagram of an Infantile Hernia. The loop of gut is behind the unobliterated funicular portion of the tunica vaginalis, and is contained within a sac formed by a process of peritoneum pushed down from the neighbourhood of the internal abdominal ring.

FIG. 156.

Diagram of an Encysted Hernia. The gut is contained within a sac formed by an invagination of the upper part of the unobliterated funicular portion of the tunica vaginalis.

said to be formed by a yielding of the adhesions at the point of closure of the funicular portion opposite the external ring. In this form of hernia the sac is invaginated into the unobliterated funicular portion, and is not placed completely behind the latter as in the infantile variety; much doubt, however, has recently been cast on the existence of true encysted herniæ by the investigations of Mr. Lockwood. Reference to the accompanying diagrams will render these various forms of herniæ more intelligible.

Interstitial hernia.—This form of hernia, called also "intra-parietal" and "hernie en bissac," is characterised by the presence of offshoots or pouches of the sac, which extend amongst the structures composing the abdominal wall. Thus, a sac may meet with some obstruction at the external abdominal ring, and may then extend either upwards, outwards, or inwards *behind* the aponeurosis of the external oblique. In other cases it passes through the external ring, but, meeting with difficulty in its further passage to the scrotum, extends between the skin and external oblique, and forms a swelling parallel to Poupart's ligament. Much more rarely the sac extends into the iliac fossa and passes between the iliac fascia and the peritoneum. This variety of hernia not infrequently complicates retention of the testis in the inguinal canal, and in such cases is the result of the obstruction offered by the testis to the descent of the gut into the scrotum.

Acquired inguinal hernia. — In the case of an ordinary acquired inguinal hernia there is no abnormal condition of the tunica vaginalis to complicate matters, and the sac is formed in the manner already described in an earlier part of the present chapter. When an inguinal hernia becomes strangulated, the seat of constriction may be either the external or internal abdominal ring or the neck of the sac, but it should be remembered that when the sac is of congenital origin its neck is almost always the cause of the constriction.

In **femoral** herniæ the protrusion takes place at the crural ring, traverses the crural canal, and subsequently appears at the saphenous opening. The sac is always an acquired one, and is not so capacious as is that of an inguinal hernia. The contents are usually part of the ileum with more or less omentum. If strangulation occur, the seat of constriction is usually outside the sac, being at Gimbernat's or Hey's ligament or the deep crural arch. On account of the sharpness of these bands of ligament, the imprisoned gut ulcerates and sloughs more

rapidly than is the case in inguinal herniæ, and it is well known to all surgeons that, on this account, the mortality after herniotomy for femoral hernia is greater than in the case of inguinal ruptures.

In **umbilical** hernia the abdominal viscera escape either through the umbilical ring itself or else through the linea alba in its immediate neighbourhood. This form of rupture is common in new-born children, and in them is readily curable by the application of slight pressure. When occurring in adult life, it is met with most commonly in very stout people with much intra-abdominal fat. In these the sac is usually of extreme tenuity, and is often gradually thinned away to such an extent that in places it becomes incomplete, the contents contracting adhesions to the surrounding parts and becoming irreducible. These contents almost always comprise omentum and jejunum, but in some cases the transverse colon is extruded. If strangulated, the seat of constriction is the aperture of exit in the linea alba, or adhesions formed by the omentum within the sac.

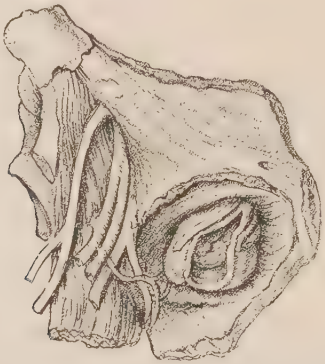
In **obturator** hernia the sac is always acquired, and is protruded through the upper part of the obturator foramen in company with the vessels and nerves of the same name. This variety of hernia is most common in thin, elderly women; the sac is always small, but sometimes causes pressure on, and pain in the course of, the obturator nerve. Strangulation is not common, and, when it occurs, the constriction is usually outside the sac and not very acute. (See Fig. 157.)

A **ventral** hernia is one in which the sac is formed by a protrusion of peritoneum at a part of the abdominal wall in which normally no aperture exists. Such herniæ sometimes occur in the middle line between the recti muscles in women who have had prolonged and difficult labours, but are more often the sequel of wounds of the abdominal wall followed by formation of a weak and yielding scar. They are very rarely strangulated.

Perineal and **sciatic** herniæ are extremely rare. In the former, the protrusion takes place between the anterior fibres of the levator ani; in the latter, through the sciatic notch, beneath the gluteus maximus.

Diaphragmatic hernia is either due to a congenital defect in the diaphragm or to injury. In the former case, the congenital aperture is almost invariably situated in the left half of the muscle; in the latter, it may be situated in

FIG. 157.



An Obturator Hernia. The sac protrudes through the upper part of the thyroid foramen, and the obturator vessels and nerve are stretched over its anterior surface.

any portion. In this form of hernia there is no true sac, the viscera escaping into the pleura, and, more rarely, into the pericardium. The viscera which escape are usually the stomach, colon, and small intestine. Strangulation is rare, but is not unknown.

Littré's hernia is the term applied to a strangulation of a Meckel's diverticulum, whilst **Richter's hernia** is the name given to the strangulation of a portion only of the whole circumference of a knuckle of intestine. To this latter condition the name of "**partial enterocele**" has more recently been given. Both these varieties of hernia are of interest, for although in neither of them is the whole

calibre of the gut mechanically obstructed, nevertheless all the symptoms of strangulation are present. It is thus evident that the vomiting and constipation which accompany strangulation are not the result of simple mechanical obstruction, but are caused reflexly by the injury to, and irritation of, the constricted peritoneum.

In connection with the subject of hernia may be mentioned **protrusions of sub-peritoneal fat** through the linea alba. They usually occur about midway between the umbilicus and the ensiform cartilage, and form soft, partially reducible swellings, about the size of a walnut or a little larger, and increasing during any straining movements or in the act of coughing. Sometimes they can be reduced and the aperture of exit in the linea alba can be clearly defined, but in all cases where they have existed more than a few months they become irreducible. I have seen these swellings in both men and women above middle age, and have had opportunities of examining two of them post-mortem. They are liable to be mistaken for true herniæ, but the mistake is of little consequence, for they are well-treated by the application of a truss.

CHAPTER LXI.

INTESTINAL OBSTRUCTION.

This term is applied to all the various forms of mechanical obstruction of the intestines within the abdomen, as opposed to the external herniæ which protrude through the abdominal walls.

The following are the chief varieties of intestinal obstruction:—Internal herniæ; volvulus; intussuception; obstruction by foreign bodies and fæcal accumulation; stricture; tumours of the intestine; pressure on the bowel from without by tumours, &c.; chronic peritonitis.

Fig. 158.

An Internal Hernia. A loop of small intestine is strangulated by a band of adhesions.

The term **internal hernia** is applied to cases in which intestine has become constricted by bands of adhesions or of omentum, or by slipping through apertures in the omentum or mesentery or into such normal openings as the foramen of Winslow and the intersigmoid fossa. Bands of adhesion are formed as the result of peritonitis, and are most common in the right iliac fossa, though they may occur in any part of the abdomen. Portions of omentum may also become adherent from peritonitis or by inclusion in the sac of an irreducible hernia. In other cases bands are formed in connection with a persistent **Meckel's diver-**

ticulum. This latter, when present, is a pouch or offshoot of the small intestine, formed by an undue persistence and development of the vitelline duct. It is situated about two

FIG. 159.

Meckel's Diverticulum. From a specimen in the Museum of St. Bartholomew's Hospital.

feet above the ileo-cæcal valve, and varies in length from an inch to a foot. In some cases it is adherent to the umbilicus from birth, and may, indeed, open onto the abdominal

wall in this situation, presenting either a cul-de-sac or else a complete tube, opening into the intestine at one end and at the navel at the other; in other cases, it contracts adhesions to the iliac fossa or to some other part of the abdominal parietes as a result probably of an attack of inflammation induced by impaction of intestinal contents; whilst very frequently it remains free and movable in the abdominal cavity. When adherent, coils of intestine may

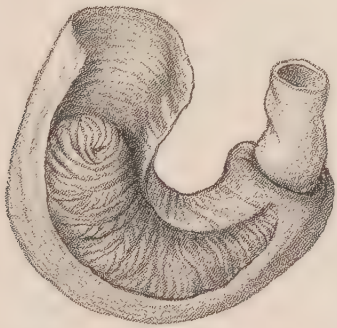
FIG. 160.

Meckel's Diverticulum opening at the umbilicus.

be caught around or compressed by it; whilst in some few instances, when free, it has been known to knot itself round the mesenteric attachment of a knuckle of bowel, although in the vast majority of cases it causes no trouble whatever unless it is attached at its extremity. Strangulation of intestine from slipping through apertures in the omentum, into the foramen of Winslow, &c., is of very rare occurrence, and does not require further comment.

Volvulus, or twisting of the intestine, is most often seen in the sigmoid flexure, but is nowhere common. In cases where the sigmoid flexure is unusually voluminous, large coils of the gut sometimes protrude almost at right angles from the course of the descending colon. The size of these coils is in most cases acquired, and appears to be the result of chronic constipation; in such cases the meso-colon is also longer than natural. If this part of the bowel be distended by fæces, it may happen that these coils become doubled over by the weight of their contents, and so

FIG. 161.



An Intussusception. The sheath has been laid open so as to show the intussuscepted portion.

twisted upon their mesenteric axis and kinked that both the passage of fæces and the free circulation of blood in the mesenteric vessels are interfered with, and strangulation results. In such cases gangrene and peritonitis rapidly ensue. Much more rarely a similar form of volvulus is found in the ascending colon, whilst the same portion of bowel has also been seen twisted around its own longitudinal axis. The cæcum also, especially when imperfectly developed, has been known to be the seat of a twist. Volvulus of the small intestine is met with only in connection with previous peritonitis and adhesion of loops of intestine; it is extremely rare.

Intussusception.—In intussusception one portion of the intestinal tube is invaginated into the lumen of that portion of intestine with which it is immediately continuous below. A complete intussusception consists of three cylinders or rings of bowel. The innermost of these is named the entering layer; the middle, the returning layer; and the external, the sheath. Intussusception is most common in children, and usually originates at the junction of the ileum with the cæcum. In this situation the ileum, with the ileo-cæcal valve, is pushed into the more capacious cæcum. If the intussusception now continues to increase, it does so at the expense of the colon, more and more of which is rolled in by the constant and excessive vermicular contraction of the intestines. It thus happens that the apex, or presenting part, of the invaginated bowel, or the "intussusceptum," throughout the development of the affection always consists of the same portion of gut which originally became intussuscepted, namely, the ileum and the ileo-cæcal valve, and thus in some cases the latter may present at the orifice of the anus. This form of intussusception is named **ileo-cæcal**, but another and much more rare form, named **ileo-colic**, is met with, in which the ileum becomes inverted *through* the ileo-cæcal valve, and the intussusceptum increases by protrusion of additional portions of ileum. Intussusceptions are also found in the small intestine and in the descending colon and sigmoid flexure, but seldom attain a large size. All true intussusceptions may easily be distinguished from similar invaginations which are formed sometimes during the irregular movements of the intestine which occur not uncommonly just before death. These latter intussusceptions are always small, frequently multiple, easily reduced, and the gut is not congested.

If an intussusception remain unreduced, congestion and subsequent sloughing of the imprisoned bowel is apt to ensue. In some cases an intussusception persists for a considerable time without serious mischief resulting, but

such instances are comparatively rare, and much more commonly the circulation in the intussuscepted gut is quickly interfered with by the traction on its mesentery. The intussusceptum swells from venous engorgement, and very soon blood escapes from the distended vessels into the interior of the intestine, as well as into the wall of the gut itself. The swelling causes, in its turn, a greater constriction of the intussusceptum where it enters the sheath, the arterial circulation is arrested, and gangrene results. In its early stages an intussusception may be reduced, but later on this becomes impossible. The irreducibility is in great part the result of the swelling already mentioned, but, in addition, adhesions soon form between the peritoneal surfaces of the entering and returning layers, as well as between the sheath and the entering layer, and fix the latter in its abnormal position. In almost all cases there is an extension of inflammation from the inflamed or sloughing intestine to the neighbouring peritoneum, and peritonitis results, whilst sometimes the sheath itself becomes ulcerated, or even gangrenous. Spontaneous cure is more rare than is generally supposed; in such cases the gangrenous intussusceptum is cast off as a slough, and the patient recovers with an intestine which is shortened in proportion to the length of that piece which has sloughed, the continuity of the canal being established by the adhesions which form between the sheath and the entering layer.

It is not easy to assign a definite cause to each individual case of intussusception, but it is probable that all owe their origin to irregular and excessive contractions of the bowel, such as occur in colic or result from the presence of any hard or foreign body in the intestine. In some rare cases the trouble may be traced to the presence of a polypus, which, being gripped by the bowel below its seat of attachment, is drawn in, together with the gut from which it grows, during peristalsis.

Obstruction by foreign bodies and fæcal accumulations.—It is not often that intestinal obstruction results

from the presence of foreign bodies, unless in this category hardened fæces be included. The various foreign bodies which are so frequently swallowed are seldom of sufficient size to block the intestine, whilst yet small enough to safely pass the œsophagus and stomach. They may, it is true, lodge in the cæcum or some other part of the alimentary tract, and cause great trouble by setting up ulceration, with the subsequent formation of fæcal abscesses or fistulæ, or may induce peritonitis; yet, withal, they seldom entirely obstruct the calibre of the intestine. A more frequent cause of obstruction is a gall-stone, and it is probable that, when it is of sufficient size to become impacted, it has gained the cavity of the bowel rather by ulcerating from the gall-bladder into the duodenum than by passing along the gall-duct, for it is difficult to conceive that a stone which would traverse the latter could block the gut. But whether the foreign body be a gall-stone or something swallowed by the patient, it is more likely to be arrested at the junction of the small with the large intestine than elsewhere, on account of the opposition offered to its further progress by the ileo-cæcal valve. Intestinal calculi, or enterolithes, are of rare occurrence; they are usually composed of phosphatic deposits formed around some foreign body, or of masses of hair or fibre; these latter are generally met with in hysterical subjects or lunatics. In other cases the calculi are composed of vegetable matter, or of masses of insoluble salts, such as magnesia, which have been taken medicinally. Any of these forms of enterolithes may attain great size, and completely obstruct the bowel.

Fæcal accumulation is the result of long-continued and neglected constipation, and is seldom seen in early life. The accumulation is necessarily gradual, and is most common in the cæcum, sigmoid flexure, and rectum. In such cases the intestine, which is already feeble and slow to contract, becomes still more paralysed by the distension and pressure of the contained mass, and, should the condition

remain unrelieved, complete cessation of all peristalsis results. In addition to the mechanical blocking of the bowel, the fæcal mass, which becomes harder and dryer the longer it is retained, causes inflammation and ulceration of the intestine, and, subsequently, localised chronic peritonitis, the gut above the obstruction becoming also immensely distended. In other cases fæcal masses not large enough to occlude the intestine remain in the cæcum and keep up persistent irritation, perhaps for months.

Stricture of the intestine.—Except in the rectum, stricture is rare, and the forms of narrowing in this part of the bowel will be dealt with more fully in the chapter on Diseases of the Rectum. The simple strictures of the intestines are, like those of other mucous canals, caused by the contraction of scar tissue at the seat of previous ulceration, those ulcers which are most often followed by such contraction being the dysenteric, the catarrhal, the tubercular, and the syphilitic. The strictures caused by the two first of these are most common in the large intestine; those by the two latter in the lower part of the ileum and the cæcum.

There is also another variety of stricture which is not due to contraction of healed ulcers, but is of congenital origin, and is the result of a contraction of the bowel at the seat of its communication with the omphalo-mesenteric duct. It has already been mentioned that in some cases this duct persists as a diverticulum, but in others the reverse condition occurs, and the process of shrinkage and obliteration, which ought to be limited to the duct alone, extends to the intestine and causes a stricture, which, in an adult, would be found about two feet from the ileo-cæcal valve.

Tumours of the intestine (not including the rectum).—Both innocent and malignant tumours are met with in the intestine, the latter being unfortunately the more common.

Of the innocent growths, the simple glandular polypus is

of most frequent occurrence. It is seen chiefly in children, and is sometimes multiple. Its common site is the lower part of the ileum and the colon. It is seldom large enough to obstruct the bowel, but, as already mentioned, may cause intussusception. Fibrous, nævoid, and fatty growths are very rare.

Malignant tumours are much more common in the large than in the small intestine. They are almost invariably carcinomatous, and frequently belong to that variety of malignant growth to which the name of adenoid cancer has been given. These growths become more common the nearer we approach to the anus, but are more frequent in the cæcum than in the ascending colon. They cause much induration and narrowing of the intestine, with the formation of a so-called "cancerous stricture.' The growths themselves ulcerate, and may furnish a discharge of blood-stained and foul fluid from the anus. Above the stricture the gut becomes dilated, inflamed, and often ulcerated, so that perforation of the bowel and acute purulent peritonitis frequently induce a fatal termination. On post-mortem examination, it is by no means rare to find no secondary growths in any of the viscera, and in a considerable number of cases there is no glandular affection. In some cases the growth is tuberous, and protrudes into the cavity of the bowel ; in others, it forms a hard ring in the wall of the gut itself.

Lympho-sarcomata are also rarely seen, and occur as multiple, soft, fleshy tumours, of a dirty-white colour, covered by mucous membrane, and especially liable to develop in Peyer's patches.

Pressure on the bowel from without by tumours, &c.—This is not a common cause of obstruction, for the bowel, being in most parts movable, is not readily compressed. Cancerous growths, from their tendency to fix and adhere to the tissues, more often cause obstruction than innocent tumours, and as examples may be quoted cancer of the pancreas, spleen, kidney, and liver. In other

cases, uterine, ovarian, or prostatic tumours compress the bowel, and in yet another class the obstruction is caused by the pressure of pus. Thus, I have seen a case in which an abscess which formed in Douglas's pouch as the sequel to rectal stricture and ulceration caused such pressure on the bowel as to induce death by obstruction, and other similar cases have been recorded. Occasionally, the glandular tumours which result from "tabes mesenterica" attain a sufficient size to compress the bowel, to which, in addition, they may contract adhesions.

Chronic peritonitis.—In tubercular disease of the peritoneum it is by no means uncommon for the coils of intestine to become matted to one another and to the enlarged glands which are often present. In consequence of this matting, a chronic form of obstruction may supervene. The condition is most common in young people. In another class of cases the peritoneal inflammation is dependent upon disseminated new growth. This is most frequently seen in connection with malignant ovarian disease, but in some cases the growth is primary in the serous membrane.

CHAPTER LXII.

DISEASES OF THE RECTUM.

IMPERFORATE ANUS.

Imperforate anus results from imperfect union between the rectum above and the posterior part of the cloaca common to the uro-genital aperture and the hind gut below.

The deformity exists in varying degrees. In some cases there is an anal depression, at the bottom of which a membrane forms a septum separating the anus from the rectum, or else the anus itself is occluded by a similar membrane, which extends backwards from the scrotum to the coccyx. In other cases, again, the anus and rectum are continuous, but the gut is much narrowed at point of their junction, and occasionally the anal orifice is obstructed by a longitudinal fold of skin extending from the coccyx to the scrotum, and bisecting the anal aperture.

In more severe examples the anal aperture is separated from the rectum by fibrous tissue an inch or more in depth, whilst in some instances the rectum itself is completely undeveloped. Not infrequently the rectum opens into the genito-urinary tract—in the female into the posterior wall of the vagina, and in the male into the prostatic urethra; more rarely, the opening is into the bladder itself. Those cases in which there is no passage for fæces terminate fatally unless subjected to surgical treatment, the infant dying from vomiting and exhaustion, or from rupture of the cæcum or colon caused by over-distension.

HÆMORRHOIDS.

Hæmorrhoids, or piles, constitute one of the most common of the diseases to which the rectum is liable. A hæmorrhoid is essentially a dilated or varicose vein, or more often a collection of such veins. According to their situation relative to the anal orifice, piles are called external or internal, but, although it is not uncommon to find internal piles existing alone, external piles are almost always associated with the internal variety.

External piles are covered by skin, and are generally multiple. When not inflamed, they give rise to but little trouble, and present themselves as swollen tabs of skin around the anal orifice. When inflamed, they form purple, shiny, tense, rounded masses, causing much pain and rectal tenesmus. If removed, they are found to consist of a looped varicose vein surrounded by fibrous tissue, the quantity and denseness of which depend upon the frequency with which the pile has been the seat of inflammation. Very commonly, the blood in the varicose vein clots when the latter becomes inflamed, and occasionally suppuration or sloughing terminates the existence of a hæmorrhoid.

Internal piles vary in structure, for, although all are composed of dilated vessels, the arrangement and character of the latter are not always identical. They may conveniently be divided into (*a*) capillary, (*b*) venous, (*c*) arterial.

Capillary piles are small, sessile swellings, with a papillated, mulberry-like surface which readily bleeds. This hæmorrhage, which is common in all cases of internal piles, is readily accounted for when we consider that the latter are covered by thin mucous membrane only, and not by skin, as are the external ones, and that, in addition, they are exposed to the pressure and irritation of the hardened fæces of the rectum. Children are but seldom the subjects of piles, but when such is the case it is the capillary pile which is almost always found.

Venous piles are the most common of the internal hæmorrhoids. They are usually multiple, and extend from just inside the anal margin to a distance of one or two inches up the rectum. They form irregular pedunculated swellings, purple in colour, soft in consistence, and not so prone to bleed profusely as the capillary variety. On section after removal, they are seen to be composed almost entirely of irregular varicose veins, surrounded by but little fibrous tissue, and a few small arterioles.

Arterial piles are comparatively rare. They are generally

FIG. 162.

The Inner Surface of a Rectum with some Large Internal Piles.

large, distinctly pedunculated, smooth and slippery on the surface, and readily bleed *per saltum* if scratched. They contain arteries of considerable size, as well as varicose veins.

The **causes** of hæmorrhoids are numerous, but, in general terms, it may be said that they are induced by anything which impedes the return of blood from the rectum. It should be remembered that most of the rectal veins open into those of the portal system, and that, as the latter have no valves, the blood-pressure at the most dependent parts of the rectal vessels is necessarily considerable.

In addition, the veins of the rectum run in a submucous tissue which is more than usually lax and yielding—a condition, which, although necessary on account of the constantly varying distension of the rectum, nevertheless gives but little support to the veins. Chronic constipation, which is one of the most fertile causes of piles, induces them in more ways than one. First, the fæcal masses compress the veins directly, and, secondly, the thickening of the muscular coat of the rectum, which often results from its distension, causes an increased difficulty in the flow of blood from the veins of the submucous tissue.

But the pressure on the rectal veins is often caused by diseased conditions of the rectum itself, and it should always be remembered that almost any disease of the rectum may be complicated by piles; this is a fact that has more than a pathological importance, for, if it is not recognised, the more serious mischief may be overlooked and an operation be undertaken for the cure of piles when the latter are perhaps but a complication of a rectal stricture or new growth. In some cases pressure on the rectum may be caused by a pregnant uterus or a uterine fibroid, and in old men by an enlarged prostate. The impediment to the portal circulation caused by a congested liver, often the result of excessive indulgence in food and drink, and still more that caused by cirrhosis of the liver tends to cause piles, whilst, in addition, the conditions which produce a more general congestion of the venous system, such as diseases of the heart and lungs, also exercise some influence.

Lastly, sedentary occupations and a feeble state of health with loss or impairment of healthy muscular tone, such as is sometimes induced by residence in hot climates, must be mentioned as causes of hæmorrhoids, and it is probable that heredity is not without some influence, for in not a few cases hæmorrhoids occur in several members of the same family.

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ISCHIO-RECTAL AND ANAL ABSCESS AND FISTULA IN ANO.

The abscesses which are common in the neighbourhood of the rectum and anus fall naturally into two divisions—those which are superficial and those which are deep. To the former the name of **anal** abscess is often applied, for they develop in the folds of skin which surround the anus. They never attain any great size, and readily burst externally; if they burst internally as well, the most common place for the formation of the internal opening is the line of separation between the external and internal sphincters—*i.e.*, just within the anus itself.

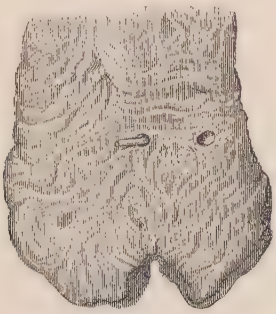
The deep or **ischio-rectal** abscesses are formed in the fossa of that name. They are situated beneath the deep fascia, and show but little tendency to come to the cutaneous surface, meeting with less resistance in their extension toward the bowel, into which they often burst above the upper margin or the internal sphincter—*i.e.*, about an inch and a quarter to an inch and a half up the rectum. Their formation is accompanied by much brawny swelling, with induration and pain.

Both anal and ischio-rectal abscesses may result from injury or exposure to wet and cold. Usually the injury is applied from without, but the presence of hard and sharp foreign substances in the rectum provides another source of traumatism, for by their passage through the mucous membrane they may allow of the escape of minute quantities of fæcal matter into the cellular tissue, and thus promote suppuration. The anal abscesses appear to owe their commencement in many cases to the irritation caused by want of cleanliness in this region.

A **fistula in ano** is the result of an unhealed abscess, and just as there are two kinds of abscesses, so are there two kinds of fistulæ. It must not be supposed that all abscesses in the neighbourhood of the rectum refuse to heal, for such is not the case, but, as a very large number

of them lead to the formation of fistulæ, it is evident that some common cause or causes must be present in all cases. One of the most constant of these is the perpetual movement of the parts, for not only are the walls of the abscess drawn up during defæcation or the passage of flatus, but the perineum is kept in motion by any violent respiratory act, and especially by coughing. Another fertile source of fistula is the passage of fæcal matter, and the irritation which is thereby set up whenever the abscess-cavity com-

Fig. 163.



The Inner Surface of a Rectum with Two Small Tubercular Ulcers, into one of which a portion of glass rod has been passed.

municates with the bowel. The association of fistula with phthisis finds an explanation in the presence of tubercular ulceration of the rectum in this disease. The ulcer, which is generally ragged, with undermined edges, permits the escape of fæcal matter, as already mentioned, and causes suppuration around the gut. Lastly, as in the case of piles, it must constantly be kept in mind that a fistula may be but a complication of some other rectal disease, such as simple or cancerous stricture, syphilitic ulceration, fissure, &c.

Three chief **varieties** of fistula are described—the complete, the blind external, and the blind internal. A **complete** fistula is one which opens both externally

through the skin and internally into the bowel; strictly speaking, this is the only form of true fistula, the others being sinuses. A **blind external** fistula is one which opens externally alone, but is "blind" towards the rectum, and, conversely, a **blind internal** fistula has an opening into the rectum alone, and none through the skin. The external opening is usually close to the anus, but in some cases is situated at a distance of several inches, or, in rare instances, as far away as the great trochanter. In healthy subjects this opening is small, and from it protrudes a small mass of healthy florid granulations. In phthisical patients the aperture is often large and ragged, the skin undermined and purple, and the base devoid of healthy granulations, and secreting a little watery pus.

The position of the internal opening depends chiefly on the kind of abscess which has preceded the fistula. If an anal one, then the aperture is just inside the anus, between the external and internal sphincters; whilst if an ischio-rectal one, the opening is placed above the internal sphincter, and is sometimes as much as three or even four inches from the anus. The character of this orifice differs, as does that of the external one, according to the constitutional condition of the patient. The fistulous track itself is lined at first by the granulation tissue of the shrunken abscess-cavity, but in cases of long standing the wall becomes thickened by fibrous tissue, and the canal is lined by a dense cicatricial membrane, showing no tendency whatever to heal.

A fistula chiefly causes trouble by the discharge of matter and the incontinence of flatus and fæces, but, if it be neglected, further mischief will ensue. On account of the occasional blocking of the track, pus is liable to be retained at times, fresh abscesses form, and either burst again through the old opening or else point in fresh places. In this way other fistulous tracts are produced, which in many cases form branching channels leading from the original sinus. As this trouble is continuous so long as it is allowed

to remain untreated, additional openings form in the bowel, the mucous membrane is destroyed by ulceration, and the submucous and muscular coats are infiltrated by fibrous tissue and other inflammatory products. In this manner the lower part of the bowel is converted into a tough fibrous tube, incapable alike of properly retaining fæces and of transmitting them, for the sphincters cease to act, not only because they are partly destroyed, but also because the mucous surface no longer maintains its normal sensi-

FIG. 164.

A Rectum with numerous Fistulæ and much Fibrous Thickening.

bility, and thus the presence of fæcal matter is not recognised by the patient, and does not excite the natural reflex contraction of the muscles. On account of the contraction of the newly-formed fibrous tissue, much narrowing may ensue, and thus lead to the development of a stricture. Some few cases terminate fatally from exhaustion, caused by profuse suppuration; others, from acute peritonitis consequent on the perforation of an ulcer through some part of the rectum covered by peritoneum.

ANAL FISSURE.

In fissure of the anus there is, as the name implies, a crack or fissure at the anal orifice. In very many cases no

other cause is apparent beyond chronic constipation, and it is probable that the disease generally results from over-distension by hardened masses of fæces. In some instances the fissure is slight and readily heals even without treatment, but usually it extends, and shows little tendency to spontaneous cure. The common situation of a fissure is the posterior margin of the anus; it usually reaches about half an inch or more up the bowel, and an examination may show that it is complicated by the presence of either piles or a polypus. It is of some importance to recognise such a complication, for, if the growth be not removed, the treatment of the fissure is liable to prove abortive. Fissures cause much pain, which is especially severe after defæcation, and it is supposed that both this symptom and their slowness to heal are alike accounted for by the exposure of nervous twigs in the floor of the fissure.

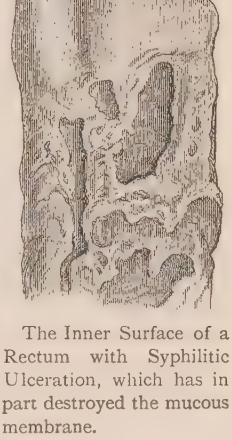
ULCERS OF THE RECTUM.

Simple ulceration of the rectum is usually of traumatic origin, and is the result of the presence of hardened masses of fæces or of foreign bodies in the rectum. The ulcer is usually single, and may be situated at any part of the circumference of the bowel. It is seldom larger than a shilling, and is of no regular shape; its base and edges are neither indurated nor unduly prominent. As already mentioned, ulceration of the rectum may lead to the formation of a fistula, and the fistula, in its turn, may be followed by an ulceration which extends up the rectum, and sometimes involves the mucous membrane in the greater part of its length.

Syphilitic ulceration is more common in women than in men, and is usually a tertiary affection. It may, however, be said at once that cases are attributed to syphilis in which there is no evidence of this disease, and it is probable that many instances of so-called syphilitic disease are really examples of simple ulceration extending up the gut, as already described under the heading of Fistula.

In typical syphilitic disease the mucous membrane of the rectum is thickly studded with small oval or rounded ulcers, the size of a pea or a lentil. These have at first but little induration of their bases, and the edges are sharply cut, and not overhanging like those of a tubercular ulcer. By their increase in size, these ulcers coalesce, and thus the mucous membrane is destroyed over an area of irregular shape. The infiltration of the rest of the rectal wall by the products of inflammation results after a time in the production of fibrous tissue, and is followed by induration and contraction of the calibre of the gut. The ulceration tends to spread up the bowel, and in this way there is first a destruction of the mucous lining and then a formation of fibrous tissue, which may in time implicate the rectum in its whole length. The discharge from the ulcerated surface is often very profuse, and in some cases the ulcers penetrate deeply into the surrounding parts. In consequence of this penetration, fistulæ are liable to form, and in some case extend into neighbouring organs, such as the vagina or the bladder. If an ulcer penetrates the rectal wall where the latter is covered by peritoneum, acute suppurative peritonitis quickly ends the patient's life. In other cases death results from exhaustion. In connection with this subject, it may be mentioned that in secondary syphilis the anus is very frequently the seat of condylomata or mucous tubercles such as have been more fully described in the chapter on Syphilis.

FIG. 165.



The Inner Surface of a Rectum with Syphilitic Ulceration, which has in part destroyed the mucous membrane.

Tubercular ulceration of the intestine, and the part it plays in the production of fistulæ, have already been

described, and it now only remains to mention briefly the other and rarer causes of ulceration. These are chiefly **dysentery** and **catarrh**, for the ulceration which is the almost constant accompaniment of new growths is better dealt with in connection with tumours of the rectum. Dysenteric ulceration is often very chronic, and may persist for years. The scars left by it are frequently pigmented. In rare cases it causes penetration of the rectal walls, with the formation of fistulæ. It is peculiarly liable to be followed by abscess of the liver.

STRICTURE OF THE RECTUM.

Rectal stricture results either from inflammation and ulceration or from the presence of new growths. Any of the varieties of ulceration described above may result in the formation of a simple stricture, which, in addition, may ensue upon pelvic cellulitis or other forms of inflammation connected with the female genital organs. In some cases stricture close to the anal orifice is dependent upon imperfect development of the communication between the bowel and the skin surface. Occasionally, stricture results from the too free removal of skin in operations upon hæmorrhoids.

Stricture occurs in all parts of the rectum, and may be either annular or tubular. In the former there is a narrow ring-like contraction of the gut; in the latter, the rectal walls are infiltrated in a considerable part of their length. In many cases in which there has been extensive ulceration, and the stricture is of long standing, the lowest portion of the gut is found, when examined by the finger, to be extremely rough and irregular, the walls being exceedingly hard and resistant, and the bowel fixed to the surrounding parts. The rectum above the stricture becomes greatly hypertrophied and dilated on account of the obstruction to the passage of fæces, and, on account of the retention of the latter, is liable to become inflamed and ulcerated. The ulceration, in its turn, is followed by

contraction of the scar tissue, and thus the stricture extends.

The ulcerated and dilated condition of the gut above the obstruction affords a ready explanation of some of the most typical symptoms of stricture. It is common in these cases to have a history of alternating attacks of constipation and diarrhœa. The constipation is the result of the accumulation of fæces above the stricture, and it is the presence of the fæcal masses which causes irritation and catarrh of the bowel, resulting in discharge of the accumulated fæces mixed with, and liquefied by, the secretion of the intestine. The ulcerated surface also supplies a discharge of pus and mucus, which collects above the sphincters, and causes frequent desire to defæcate, the patient passing a mixture of pus and blood. When the stricture is near the anus, the motions are moulded by it rather than by the anal orifice, and become narrow and pipe-like; but if the obstruction be in the upper part of the rectum, the fæces collect again below the stricture, are moulded by the muscular rectal walls, and finally shaped by the anus, so that any narrowing which has been produced in the passage through the stricture is lost in the rectum below it.

Stricture of the rectum may end fatally in more ways than one. In some cases the discharge from the ulcerated surface causes death by exhaustion or amyloid disease; in others, additional trouble results from the implication of the bladder and the formation of a fistulous communication; sometimes peritonitis is caused by the penetration of an ulcer high up the bowel; and lastly, the opposition to the passage of fæces may culminate in complete intestinal obstruction.

TUMOURS OF THE RECTUM AND ANUS.

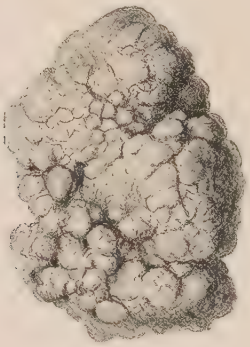
Tumours of the rectum, like those of other parts, are either innocent or malignant; the former will be first described.

Papillomata are sometimes, though not frequently,

met with around the anal orifice. They present the same appearances as similar growths elsewhere, being roughened on the surface, not indurated nor infiltrating the surrounding tissues, and often discharging a watery, blood-stained fluid. They are usually small, but occasionally attain considerable proportions. They occur chiefly in adults.

Papillomata, or villous tumours, are also found in the rectum, although they are not common in this situation. They vary in size, and I have seen one as large as a clenched fist, though they are more commonly not much larger than a walnut. They are bright red in colour, and

Fig. 166.



Papilloma of the Rectum.

their surface is covered with large rounded papillæ ; their base of attachment is broad. Although very vascular, they do not cause much hæmorrhage from the bowel, but are characterised clinically by the constant escape of a profuse watery discharge, which the patient is often unable to retain. I have seen one case in which the base of the papilloma was continuous with a cancerous growth of recent origin in the rectal wall (see Fig. 166).

Polypi are found at all ages, but are relatively common in children. They are either (*a*) glandular or (*b*) fibrous. The **glandular** or adenomatous polypi are usually about

the size of a small hazel-nut, are attached by a stalk or pedicle which is often an inch or two in length, have a papillated surface, and are prone to bleed. In rare instances they are multiple, and may be numbered by hundreds, extending up the rectum as far as the finger can reach and bleeding profusely. On microscopical examination, these glandular growths are found to consist of numerous

FIG. 167.

A Rectum with Multiple Glandular Polypi.

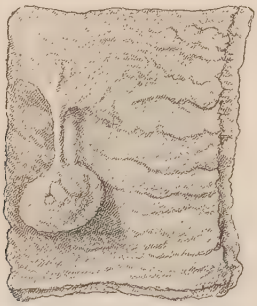
Lieberkuhn's follicles closely set in a matrix of loose fibrous tissue.

The **fibrous** polypi are not so common as the adenomatous ones. Their surface is smoother, their pedicles shorter, and their tendency to bleed not so marked as in the case of the glandular polypi. Sometimes they attain a very considerable size, and there is a specimen in the museum of St. Bartholomew's Hospital which was removed

by my friend Mr. Everley Taylor from a girl of about twenty, and weighed no less than a pound. Such cases are, however, extremely rare. On microscopical examination these polypi are seen to be composed of loose connective tissue; sometimes they are very soft, and contain much serous fluid in the connective-tissue spaces.

Dermoid tumours of congenital origin, and polypoid in shape, have been occasionally met with in the rectum; they are sometimes covered by long silky hairs.

FIG. 168.



Portion of a Rectum with a Pedunculated Glandular Polypus.

Nævi are very rare, but are liable to be dangerous to the life of the patient by reason of the copious hæmorrhage which they occasionally cause. Like similar growths in other parts, they are met with in children.

Malignant tumours of the rectum are practically always carcinomata, for the sarcomata are so rare that they do not require separate mention. But, although all the malignant growths are of epithelial origin, they are not all alike either in their physical properties or microscopical structure, and may conveniently be divided into two classes—(*a*) the adenoid or glandular cancers and (*b*) the scirrhous cancers.

The **adenoid carcinomata** have been shown by Mr. Cripps to form the greater number of the malignant

tumours of the rectum. They may grow in any part of its length, but are of more frequent occurrence in the lower than in the upper part of this portion of the bowel. They grow from the mucous membrane, and show a great tendency to extend into the lumen of the gut. At first they are covered by the mucous membrane, and form soft, partially pedunculated, smooth masses, movable on the deeper part of the rectal walls. As they increase in size,

Fig. 169.

The Inner Surface of a Rectum with an ulcerated mass of Adenoid Carcinoma.

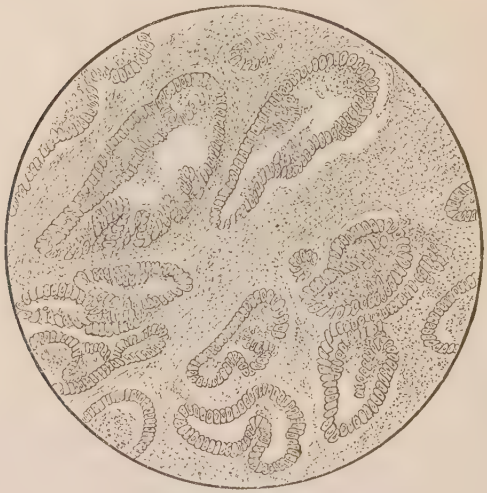
their surface becomes ulcerated and discharges a mixture of blood and mucus, their deeper portions grow into, and gradually implicate, the whole rectal wall, and, chiefly as the result of irritation, they become more firm and indurated. Their growth is not rapid, and may extend over periods of even four or five years or more. Sometimes they implicate neighbouring organs, and cause fistulous openings into the bladder or vagina, or on to the neighbouring cutaneous surface. In some instances they neither infect the lymphatic glands nor cause secondary growths, but at other times the reverse is the case, and the liver more than the other viscera is liable to metastatic deposits.

All malignant growths cause a narrowing of the calibre

of the gut, and produce the so-called malignant stricture of the rectum.

On microscopical examination, adenoid cancers are found to be composed of a loose fibrillar stroma, in which are embedded numerous tubular glands lined with columnar epithelium, as well as masses of epithelial cells lying loose in alveoli, and not forming any definite glandular structure. In these tumours, indeed, there may often be seen all

FIG. 170.



Section of an Adenoid Carcinoma of the Rectum showing the irregular cavities lined by columnar epithelium. (Zeiss, A.)

gradations of structure between a definite adenoma and a typical carcinoma, and, as a result, there are many irregularly formed and imperfectly developed gland-tubules. It is not always easy to say at first sight whether a given growth is a simple adenoma or an adenoid cancer, but the point may be settled by noticing whether or not the growth implicates the submucous and muscular coats. If it does so, then the tumour must be looked upon as malignant; whereas if it is only a surface growth, not

infiltrating the rectal wall, it must be considered a simple glandular polyp.

PROLAPSUS ANI ET RECTI.

The former of these terms is applied to an abnormal protrusion or eversion of the mucous membrane of the rectum through the anal orifice; the latter, to an eversion of the whole thickness of the rectal wall. The mucous membrane of the rectum is normally so loose that it protrudes slightly during defæcation, and anything which causes excessive straining will result in excessive protrusion or prolapse. The commonest cause of prolapse of the anus or rectum is intestinal irritation caused by the ingestion of unsuitable food, and we thus find the disease most often in unhealthy, ill-fed, flabby, and rickety children. Any disease of the rectum may be complicated by prolapse, but those which most often induce the trouble are rectal polypus, piles, and thread-worms. Prolapse is also liable to result from any disease of the urinary organs which induces straining, and is common in children suffering from phimosis or calculus.

The extent of the prolapse varies greatly; in some cases only a small fold of mucous membrane protrudes, whilst in others several inches of the lower part of the rectum projects as a tube from the anus; the prolapse tends to increase so long as its cause remains. At first the mucous membrane is everted only during straining, but as time goes on the sphincters become lax and atonic, the attachment of the mucous membrane to the submucous tissue less and less firm, and, finally, the bowel is more often prolapsed than not. The result of the constant exposure is inflammation of the mucous lining, accompanied by a catarrhal discharge, and in cases of long standing this may be succeeded by ulceration or even sloughing of the prolapsed parts, the latter being usually the result of a strangulation of the protrusion by the anal orifice.

PRURITUS ANI.

This is a term applied to a sensation of itching and irritation of the anus and surrounding skin. It sometimes complicates piles, or may result from the presence of rectal worms, but is most often caused by overfeeding and its accompanying constipation and congested liver; it is also common in gouty subjects. As a result of the irritation and consequent scratching, the skin may become eczematous.

CHAPTER LXIII.

PERITONITIS.

Inflammation of the peritoneum may be either acute or chronic, diffuse or localised. The acute inflammations are usually also diffuse, but the chronic are not necessarily localised, although this is frequently the case.

Acute diffuse peritonitis is a septic, spreading inflammation of the peritoneum. It may result from such causes as penetrating wounds of the abdominal wall; extension of inflammation from neighbouring parts—*e.g.*, from the cellular tissue in cases of urinary extravasation; rupture of the stomach or bowel, or their perforation by ulcers, tumours, or foreign bodies, and the escape of the intestinal contents—so-called "perforative peritonitis;" rupture of abscesses into the peritoneum, especially of perityphlitic abscesses; gangrene of the gut following strangulation, or any of the varieties of intestinal obstruction; and septic conditions of the system, such as septicæmia and pyæmia.

The commencement of peritonitis is marked by a great distension of the vessels, and a consequent reddening of the inflamed part. This is usually very noticeable, and at once attracts attention when the abdomen is opened at a post-mortem inspection. As the inflammation progresses the redness becomes less noticeable, the surface appears dull, and on closer examination this alteration in colour is found to be the result of exudation, which covers the inflamed parts with a thin layer of fibrin, and causes the surface of

the intestine to assume a finely granular or ground-glass appearance. Very soon, as the exudation increases, the serous surface becomes flocculent, and shreds of fibrin in the form of a fine network may be seen crossing each other in an irregular fashion. The fibrin also causes adhesion of the folds of intestine, so that the latter are found to be stuck together and partially fixed to the parietal peritoneum. The intestines themselves are greatly distended, and contain large quantities of gas—a condition which results from paralysis of their muscular coats.

In the later stages of septic peritonitis, the serous cavity is more or less filled by fluid, which at first is serous and blood-stained, but soon assumes a flocculent appearance, with shreds of fibrin floating in it, and finally becomes purulent. The fluid collects in the most dependent parts, and especially in the pelvis, but it is also prone to become localised by adhesions formed between the intestinal folds, and may thus form collections shut off in parts from the general peritoneal cavity. The intestinal walls are also swollen by exuded fluid, and softened so that they are easily lacerated; in some cases the distended capillaries give way, and cause small sub-serous petechiæ or ecchymoses.

Microscopical examination shows that in the early stages of the affection there is an exudation of serum and leucocytes, and a separation of the endothelial cells which line the serous membrane. As the inflammation progresses both red blood-cells and the fibrin-forming elements of the blood exude, and the coagulated fibrin is deposited on the inflamed surface or floats in flakes in the exudation. More and more leucocytes make their appearance, and soon the fluid is so laden with them as to become sero-purulent or purulent. Sometimes free gas, the result of decomposition, is found in the abdominal cavity. In acute diffuse peritonitis the conditions described are found in all parts of the serous membrane, but where, as is usually the case, the inflammation has extended from

some definite place, there all the signs will be found more accentuated and advanced than elsewhere.

Acute, diffuse peritonitis, in the absence of treatment, is almost invariably fatal. It is accompanied by severe collapse, with persistent sickness and constipation, and the general symptoms of septic poisoning. In otherwise healthy adults there is usually severe pain, and, at first, pyrexia; but in old and feeble patients there is often but little pain, and the temperature is frequently sub-normal.

Subacute and chronic, localised peritonitis commonly results from an extension of some inflammatory process in or around one of the viscera. Thus, it is frequently caused by inflammation of the cellular tissue around the cæcum, of the uterus or ovaries, of the bladder in cases of chronic cystitis, &c. It is found in the neighbourhood of chronic ulcers of the stomach and duodenum, and around the liver in cirrhosis. In another class of cases it results from the irritation of new growths—*e.g.*, cancer of the liver, pancreas, stomach, &c.—or may be set up by contusion of any part of the abdominal wall.

The appearances met with in this form of peritonitis are very similar to those seen in the earlier stages of the acute form of the disease already described. The redness, however, is less marked, and the exudation of fluid is but slight. In these cases, moreover, there is a tendency to the development of fibrous adhesions, and, in place of the slight stickiness seen in the acute inflammation, there is a formation of fibrous tissue, which causes fixation of various viscera to one another or to the abdominal parietes. These fibrous bands are in some cases gradually elongated by the constant dragging of the attached tissues, and may thus at some future time be a cause of entanglement and strangulation of intestine in the manner already described in the chapter on Intestinal Obstruction. In their mode of formation these bands do not differ from the fibrous tissue which forms the scar in the case of a wound; they are developed from the exuded cells, and the

blood-vessels which at first permeate them subsequently shrivel and disappear.

Chronic diffuse peritonitis may be the sequel of habitual engorgement of the serous membrane resulting from diseased conditions of the heart, lungs, or liver; it is also met with in cases of chronic interstitial nephritis. In cases of disseminated new growth—such, *e.g.*, as results sometimes from malignant ovarian disease—chronic peritonitis, with effusion of plastic lymph and the formation of adhesions, is of common occurrence. The changes met with in the localised form of the disease are seen in the diffuse variety spread over a larger area, but thickening of the serous membrane is more common in the diffuse than in the localised affection. Another and very important cause of chronic diffuse peritonitis is tubercle. In examples of this form of inflammation, the serous membrane becomes thickly studded with grey tubercles, which in some cases are present in such numbers as almost to completely cover the whole secreting surface. There is much matting of the intestinal coils, and exudation of a considerable amount of fluid; the mesenteric glands are commonly much enlarged, and often form masses of large size, to which the intestines become adherent. Tubercular peritonitis is often part of a general tuberculosis.

CHAPTER LXIV.

DISEASES OF THE VERMIFORM APPENDIX.

Catarrhal inflammation.—Like other parts of the intestines the vermiform appendix may be the seat of catarrh, and very probably it often shares in catarrhal inflammation of the cæcum. It is, however, much less able to recover quickly from the effects of inflammation than are other portions of gut, and the attacks are very liable to recur; these facts are perhaps partially explained by the narrowness of the attachments of the appendix, and by the consequent difficulty which its veins and lymphatic vessels must find in emptying their contents, and so relieving the congestion and œdema which ensue on inflammation.

The swollen mucous membrane also tends to occlude the orifice leading into the cæcum, and thus causes the products of inflammation to be retained in the appendix, and a similar retention is in other cases due to a kinking, bending or twisting of the appendix from the dragging of adhesions around it. As in other catarrhal inflammations also the secretion from the inflamed membrane, although at first mucoid, becomes in time purulent, and thus the appendix may be distended by pus which cannot find an exit into the cæcum.

As a result of several attacks the appendix becomes permanently swollen and œdematous, so that it may be as thick as a finger, and, its peritoneal coat sharing in the inflammatory process, may contract adhesions to the sur-

rounding viscera or to parts of the abdominal wall. When placed in a natural position the appendix usually becomes adherent to the iliac fossa, the cæcum, or a coil of small intestine, but in cases where the cæcum is loosely attached by a long mesentery the appendix may pass into the pelvis, and may become adherent to the ovary, broad ligament, rectum, ureters, or pelvic peritoneum. Finally, it is probable that after a prolonged catarrh the appendix may ulcerate.

The attacks of inflammation vary greatly in intensity, and the symptoms differ in proportion, but there is always colicy pain, and usually vomiting, in addition to which there may be considerable intestinal distension and symptoms of peritonitis. The earlier attacks are usually of less severity than the later ones, and in many patients there is left a good deal of permanent tenderness, with perhaps some swelling; when this is the case the appendix will almost always be found in an unhealthy condition, and under these circumstances a relapse may occur at any time.

The severity of the symptoms which accompany inflammation of the appendix, and which seem out of proportion to the size and importance of the organ, are to be explained by the frequent extension of the inflammatory process to the neighbouring peritoneum. It has been shown that when any part of the intestine is acutely inflamed, the bacteria which exist normally in its lumen are able to penetrate the softened walls and to accumulate in the inflammatory exudation outside the gut, thus coming into contact with the surrounding peritoneum and causing peritonitis. It is now generally believed that the bacterium coli commune is the chief organism thus concerned; and it is further the opinion of some experimenters that the virulence of this bacillus is increased by various unhealthy conditions of its surrounding mucous membrane, so that in inflammation of the appendix not only is it an easy matter for the bacilli to penetrate its soft coats, but the bacilli themselves are much more capable of causing inflammation.

ULCERATION DUE TO FÆCAL CONCRETIONS AND FOREIGN BODIES.

The most common cause of ulceration of the appendix is the impaction of a fæcal concretion. Most concretions are oval or oat-shaped masses resembling a cherry-stone or date-stone, and it is probably this similarity which has given rise to the very prevalent idea that ulceration is

FIG. 171.

A Twisted Vermiform Appendix containing a Concretion.

generally due to impaction of foreign bodies. The concretions vary in consistency, and may either be no harder than firm fæces, or may feel as hard as a solid foreign body; on section these hard masses often appear laminated, and they occasionally contain cretaceous matter. A foreign body may similarly be enclosed in a fæcal mass or in calcareous material.

The ultimate results of the impaction of a fæcal concretion or of a foreign body are much the same, for either of them will cause inflammation, and, in time, ulceration

of the mucous membrane, with perhaps suppuration in the parts around.

When ulceration has commenced the neighbouring tissues soon become inflamed, and it depends partly on the position of the appendix in which direction the inflammation spreads.

First.—It may extend towards the peritoneum ; an event most likely to occur in the case of an inflamed appendix lying free amongst coils of intestine in the iliac fossa. In such a case, when the ulceration has not yet perforated the appendix, the coils of intestine very often become adherent, as a result of plastic peritonitis, and thus the appendix becomes shut off from the general peritoneal cavity by adhesion, and is itself closely attached to its surroundings by similar adhesions. If, after this has taken place, suppuration occurs around the appendix, or if the ulceration extends in the appendix and ultimately perforates it, the fæcal matter thus set free and the pus will be shut off from the general peritoneal cavity by the adherent coils of intestine, and an abscess will form, whose walls are composed in part of peritoneal adhesions, gut, and the appendix itself ; the patient being thus saved for the time from general peritonitis. If, under these conditions, the pus be evacuated by a timely incision, the usual course of the case is towards recovery, but it is evident that if left alone the adhesions are more liable to yield as the abscess enlarges than are the abdominal walls, and that at any time general peritonitis may result from extension of the suppuration. Further, it is plain that if, when a peritoneal abscess has been opened, an attempt be made to remove the appendix by separating it from its adhesions, it is very probable that the peritoneal cavity will be opened and an unnecessary risk of general peritonitis incurred.

In some cases, however, adhesions do not form sufficiently early or firmly enough to protect the peritoneal cavity, and then if pus forms around the appendix, or if the latter ulcerates or sloughs as a result of the impaction of fæces or

of a foreign body, the inflammation may at once involve the peritoneum, and general peritonitis may supervene.

Secondly.—The inflammation may extend towards the cellular tissue in the iliac fossa. In this case the pus tends to collect superficially to the iliacus muscle, and, tracking towards Poupart's ligament, involves the cellular tissue of the abdominal wall. In such cases there is generally some œdema and swelling of the abdominal wall, and at a later stage redness and increased heat of the skin, conditions which are not generally found when the inflammation spreads towards the peritoneum, and, unlike what is found in these latter cases, when the cellular tissue is involved the symptoms of peritonitis are generally absent. Collections of pus in the iliac fossa tend to point above Poupart's ligament.

When an abscess connected with the vermiform appendix points or is opened by the surgeon, it usually heals up in time with proper treatment, even if there be some fæcal matter in the discharge, and, if a fæcal fistula result from an abscess in the right iliac region it will almost always be found to communicate with an opening in the cæcum. This opening may either be due to some primary ulceration of the cæcum itself or may result from suppuration around an inflamed appendix, and in some cases of the latter class the abscess discharges its contents into the cæcum instead of in other directions.

Tubercular disease and ulceration of the appendix may result in similar complications to those just described, but are not of common occurrence.

Malignant disease of the cæcum may also be complicated by suppuration in the right iliac fossa and may so simulate simple inflammation of the appendix.

CHAPTER LXV.

DEFORMITIES, INJURIES, AND DISEASES OF THE FACE AND SKULL.

HARE-LIP AND CLEFT PALATE.

The causes of the deformities known as hare-lip and cleft palate are best explained by reference to the development of the face. The nose and mouth at first form one large cavity, which is subsequently partitioned off by growths from its roof and lateral boundaries. From the roof the "naso-frontal process" descends, and from the tissues forming it there are developed in the middle line the nose with its septum, the central portion of the lip, and the pre-maxillary bone, or that portion of the upper jaw which carries the incisor teeth. The naso-buccal cavity is thus separated into two lateral portions, which are subsequently represented on each side by the nostril and the antrum.

From the sides of the common cavity two other processes simultaneously develop, and grow inwards towards the middle line. These are named the "maxillary processes," or the "superior maxillary plates," and from them the cheeks, the sides of the lips, and the whole of the upper jaw, with the exception of the inter-maxillary bone, are formed, the cavity of the nose being now shut off from that of the mouth by the hard palate.

Both hare-lip and cleft palate result from a failure in the normal union between the naso-frontal and the maxillary processes. If that part of the latter which forms the lateral portion of the lip fails to join on one side with the

central portion of the lip, a "single hare-lip" results. If the failure occurs on both sides, the hare-lip is said to be double.

If the two maxillary plates do not fuse in the middle line of the palate posteriorly, the uvula is bifid or the soft palate cleft. If the failure to join is more complete, the hard palate is fissured, the fissure being in the middle line posteriorly, but running to one side or another of the inter-maxillary bone anteriorly, and thus is seen in front as a cleft between the lateral incisor and the canine tooth. In hare-lip the deformity may consist of nothing more than a slight notch on the mucous edge to one side of the middle line. More commonly, however, there is a greater failure, and the cleft passes through the whole depth of the lip and perhaps into the nostril. When the deformity is double, the central portion of the lip may be attached to the end of the nose.

In the slighter varieties of cleft palate, as already mentioned, the soft palate alone may be implicated, but in the more common form, where the cleft extends to the bony structures, the imperfection results in a communication between the nose and mouth, with consequent difficulty in swallowing and sucking, combined with a nasal intonation of the voice. The reason why the cleft in the hard palate is in the middle line behind, but to one side in front, is that the posterior parts of the palatine processes are formed from the maxillary plates alone, whilst the arch is completed in front by the inter-maxillary bone. The fissure behind is thus a cleft between the two maxillary plates; in front between the maxillary plate and the naso-frontal process, or that part of the latter called the inter-maxillary bone.

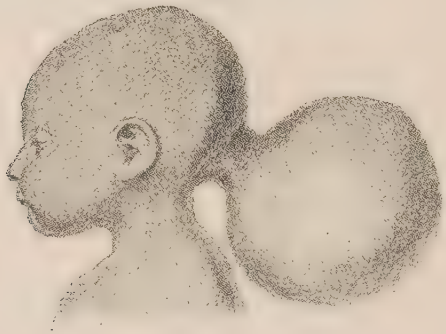
The nasal septum is usually attached below to the palatine process on the side opposite to that on which the deformity occurs. In some instances, however, the cleft is double in front, the inter-maxillary bone not being united to the maxillæ on either side, in which case the nasal septum also is unattached except to the inter-maxillary bone.

It only remains to be added that in severe examples of cleft palate there is almost always hare-lip, and that, *vice versâ*, bad cases of the latter are usually complicated by cleft palate. The incisor teeth in cases of cleft palate are commonly undeveloped or imperfectly formed.

MENINGOCELE AND ENCEPHALOCELE.

Cerebral meningocele and encephalocele are protrusions of the cerebral membranes or of the brain itself through

FIG. 172.



Occipital Meningocele.

abnormal apertures in the skull. The most common site for such a protrusion is the occipital region, just behind the foramen magnum.

In other cases the tumour appears at the anterior fontanelle; at the root of the nose, between the nasal and frontal bones; at the base of the skull, passing into the nares or pharynx; and, lastly, at the external angles of the orbit.

The membranes that are protruded are the dura mater and arachnoid, the cyst being distended by cerebro-spinal fluid. When the cyst contains brain matter, the latter is generally thrust out by distension of the ventricles with fluid. Meningocele is commonly associated with hydrocephalus.

TRAUMATIC MENINGOCELE OR TRAUMATIC CEPHALHYDROCELE.

These terms have been applied to a fluid swelling in the scalp which occasionally follows fracture of the skull in infants or very young children, and communicates with the interior of the cranial cavity.

The history of these cases is that after an injury to the skull of an infant a fluid swelling is noticed on the head, and is commonly taken to be hæmatoma. After a few

FIG. 173.

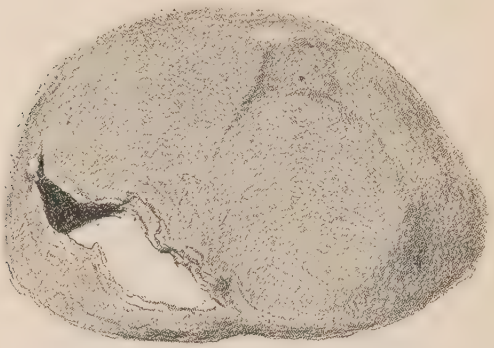
Frontal Meningocele.

days or weeks the swelling is noticed to pulsate, and increases in size. Its development is not productive of any symptoms, and if the child doos not die of coincident brain injury the tumour appears to cause no serious or fatal illness. In most cases the swelling gradually increases in size, and as it does so the definite edges of an aperture in the skull can be plainly felt. In one case I have seen there was also considerable eversion of the bone surround-

ing the cleft. In a few cases no pulsation has been detected.

Post-mortem examination shows that there has been originally a fracture of the skull with a laceration of the dura mater, and that the cerebro-spinal fluid has thus been enabled to escape into the tissues of the scalp. It is further clear that in some cases there has been also a laceration of the brain and a communication established between the

FIG. 174.



Skull showing a large Aperture in the right parietal bone in a case of Traumatic Meningocele.

external fluid swelling and the lateral ventricle of the affected side.

In all cases the original fissure in the bone rapidly increases in size, and in the case from which the accompanying drawing was made, the aperture measured three inches in length and three-quarters of an inch in width, although the injury was only ten weeks old.

The explanation offered for the formation of these swellings in the case of fractures of the skulls of infants is that, on account of the close attachment of the dura mater, this membrane is more likely to be torn than it is in adult skulls, whilst, on account of the thinness and flexibility of the skull, the bone is peculiarly liable to be driven inward,

and to wound not only the dura mater but also the brain beneath it. The rapid increase in size of the aperture in the bone is more difficult to account for, but it appears to me that it may be due to either an injury to the ossifying centre of one of the cranial bones, or may result from absorption caused by the pressure of the fluid which occupies the cleft.

DISEASES OF THE SCALP.

Although the scalp is the seat of various growths, there are not many of them which are in any way peculiar to this situation, yet some of them require special mention on account of certain conditions associated with their development in this region.

Dermoid cysts are of comparatively common occurrence in the frontal region, and close to the margin of the hairy scalp. They are always situated beneath the deep fasciæ, and form rounded swellings about as large as a nut, and seldom larger. Although congenital in their origin, they frequently do not attain a sufficient size to attract attention before the third or fourth year. In some rare cases the bone beneath these cysts is deficient, and the edges of an aperture in the skull may be felt, the tumour thus lying directly on the dura mater, and in some cases being attached to it.

Sebaceous cysts of the scalp are common in middle life and old age. They are frequently multiple, and form rounded swellings thinly covered with hair, and of various sizes. On accoun of the absence of subcutaneous tissue in the scalp, the base of such a cyst is closely attached to the aponeurosis of the occipito-frontalis, and this may easily be wounded and the loose cellular tissue beneath opened if the cyst is dissected out instead of being split open and shelled out as is generally done.

Other tumours of the scalp are nævus, cirsoid aneurysm, and soft fibroma, whilst both epithelioma and rodent ulcer are also met with in this situation.

Of the tumours of the skull itself, the most important are the ivory exostoses, and the pulsating sarcomata. The former have been already described on page 338. The sarcomata appear to originate in some cases from the dura mater, and in others from the diploë. In either case the cranium is soon perforated, and the growth becomes prominent beneath the scalp. On account of its relations to the dura mater the pulsation of the brain is readily transmitted, and is a prominent feature in these cases. Such growths are in a considerable proportion of instances multiple, and an examination of the bones of a skull so diseased, shows that the apertures made by the tumours are singularly circular and cleanly cut, looking almost like trephine-holes.

INJURIES OF THE HEAD.

The common injuries of the **scalp** do not call for any lengthened description, although there are a few points which require mention. On account of the density of the scalp, and the absence of subcutaneous tissue, wounds of this region do not readily cease bleeding, the vessels being unable to retract or contract efficiently by reason of the toughness of the tissue which surrounds them. If a scalp-wound suppurates, the pus is very liable to become diffused in the loose cellular tissue beneath the aponeurosis, although this will not occur if the aponeurosis has not been injured.

Contusions of the scalp are very liable to cause the formation of blood tumours or hæmatomas, for the cellular tissue beneath the occipito-frontalis offers little or no resistance to the escape of blood from an injured vessel, and thus the aponeurosis may become widely separated by a collection of blood beneath it. Such an effusion is called a "sub-aponeurotic cephalhæmatoma." In another class of cases blood may be extravasated beneath the pericranium, and form a "sub-pericranial cephalhæmatoma." Here the blood tumour is smaller, and is limited by the attachment

of the pericranium to the sutures, so that whereas in the first-mentioned variety the blood tumour is irregular in size and shape, and is unlimited in severe cases except by the attachment of the occipito-frontalis, in the second variety the tumour does not extend beyond the limits of the bone which has been contused; the bone in question is commonly the parietal bone, and the subjects of the injury are infants or young children. In all cases of sub-pericranial effusions, the blood-clot beneath the periosteum feels to have a very hard bone-like ridge, whilst the swelling is soft in the centre. For this reason these cases are sometimes mistaken for instances of depressed fracture, the hard ridge being mistaken for the edge of the broken bone. Hæmatomas of both varieties are usually entirely absorbed without difficulty, and require no surgical interference.

Concussion of the brain.—The post-mortem appearances of cases which during life presented the symptoms commonly recognised as those of "concussion of the brain," give but little pathological support to the use of a misleading title. The cases fall into one of three classes:—1st, contusion and laceration of the brain, with hæmorrhage into the membranes; 2nd, diffuse ecchymosis of the brain; 3rd, diffuse meningeal hæmorrhage without severe cerebral injury. Secondary complications, the result of inflammation, are dealt with on page 600.

Of these three classes, the first is undoubtedly the most common, and in at least nine out of every ten cases of "concussion of the brain" contusions or lacerations will be found on post-mortem examination. It is, of course, perfectly certain that many cases of such injury recover, and it is probable that even severe lacerations are not necessarily fatal. I have made very many post-mortem examinations of these cases, and have usually found tearing of the under surface of the frontal lobes where they rest on the irregular orbital plates of the frontal bone, or of the temporo-spheroidal lobe where it lies on the rocky mass of

the petrous bone, and have also found considerable bruising of these parts when during life there were but few symptoms, and when death had resulted from other causes. In any of this class there may be considerable hæmorrhage beneath the pia mater, or, if the latter be torn, as it usually is, into the arachnoid cavity; in many patients this hæmorrhage is the direct cause of death by means of pressure on the cerebrum and the supervention of coma.

Diffuse ecchymosis is much more rare than is localised laceration. I have seen one or two examples of it, the best marked case being that of a girl of nineteen, who died after unconsciousness of three days' duration without coma or paralysis. After death neither the meninges nor the cerebral surface showed any abnormal appearance, but on section the brain was found dotted over in all parts by numerous minute ecchymoses, the largest of which were but little bigger than a pea; they evidently resulted from the rupture of numerous minute vessels.

The third class, where the chief lesion is a diffuse meningeal hæmorrhage, is necessarily difficult to separate from the first, in which there is both laceration of the brain and effusion of blood. It is indeed evident that if blood collects in large quantities in the sub-arachnoid space or the arachnoid cavity, there must be some rupture of a vessel in the pia mater on the surface of the brain, and in these cases the brain also is commonly found lacerated by the injury or by the effused blood. There are, however, a few cases in which the brain is but little torn, and I have made a post-mortem examination of a case in which the sub-arachnoid space contained a large quantity of blood, which had been effused from a lacerated branch of the middle cerebral artery, the brain itself being practically uninjured. In cases such as this it is evident that death may result from compression of the brain, but the lesion is not necessarily fatal.

Lastly, it must be mentioned that cases are recorded in which death has rapidly followed on head injury, and no

lesion has been discovered post-mortem. It is very doubtful indeed whether such cases of cerebral concussion occur at all, and it is certain that they are extremely rare. Many of them probably are really instances of injury to the cervical spine, and of laceration of the spinal cord. I have myself twice performed post-mortem examinations on men who have died quickly after head injuries, and who were partially unconscious after the accident which proved fatal. In each case the cause of death was supposed to be concussion, and in each, when I found no lesion of the brain or its membranes, I examined the neck, and found fracture of the cervical spine high up, and laceration of the spinal cord. Had the latter not been examined, death would certainly have been attributed to concussion of the brain without the presence of any discoverable lesion.

INTRA-CRANIAL HÆMORRHAGE.

In cases of **meningeal hæmorrhage** the blood may collect in different situations, namely, (*a*) between the dura mater and the bone; (*b*) in the sub-dural space or arachnoid cavity; (*c*) in the sub-arachnoid space.

In the first variety, the hæmorrhage is commonly the result of laceration of the middle meningeal artery. This vessel is more likely to give way than are the other arteries of the meninges, because it grooves the cranium very deeply, and because the bone in question is very thin. Lacerations of these vessels are indeed almost invariably the result of fracture of the parietal or squamous bones, the artery being torn at the seat of fracture. On account of the size of the injured vessel, blood is liable to be expelled with considerable force, and thus the dura mater is gradually separated over a considerable area. The effused blood, however, can only collect at the expense of pressure upon the subjacent brain, and thus in typical cases there is a history of slowly developing drowsiness and hemiplegia, culminating after an hour or two in coma and in death. In many cases, however, the typical symptoms are masked by the symptoms of

shock and concussion, which result from the same injury which produced the fracture.

A post-mortem examination of a case of subdural hæmorrhage shows considerable swelling of the temporal fossa, caused by the escape of blood through the fissure in the bone, and a blood-clot of considerable size—often as large as half an orange—between the bone and the dura mater. Beneath this the brain is either flattened, or, in severe cases, its normally convex surface may be actually concave.

Hæmorrhage into the arachnoid cavity has been already dealt with in connection with the question of concussion of the brain, but in those cases where the lesion is not a fatal one, the extravasated blood may either become absorbed or encysted. The latter event is comparatively common, the encapsulation of the fluid resulting from adhesion of the opposed serous surfaces at the edges of the extravasation, and being caused by the irritation set up by the blood. The extravasated blood is said in some cases to undergo further change, its fibrin being deposited, its red corpuscles disintegrated, and its colouring matter being removed after the lapse of a considerable time.

But although hæmorrhage into the arachnoid cavity is not necessarily immediately fatal, the retention of the blood in the arachnoid sac may be productive of serious symptoms of cerebral irritation, and in some recorded cases of insanity. Thus, I have seen a man who was originally admitted into a London hospital for concussion, and who remained unconscious for several weeks. After three months he was discharged, but was never able to resume his work. He then began to suffer from severe headaches and frequent fits, as well as from attacks of violent and causeless passion. A year after the accident he was admitted into St. Bartholomew's Hospital, in a semi-comatose state, supervening upon numerous fits, and in a dying condition. A post-mortem examination showed a large collection of blood-stained dark fluid in the sub-dural space. On the other

hand, I have, quite by chance, found a blood-cyst in the arachnoid as large as a hen's egg, in a patient who never complained of any head symptoms at all, and in whom I could obtain no history of severe injury.

The effusion of blood in the sub-arachnoid space does not require any special description. If not effused in quantities large enough to prove fatal, it appears to be satisfactorily removed.

INTRA-CRANIAL SUPPURATION.

Suppuration within the skull may result from injury under various circumstances.

First, it may follow on the symptoms of concussion, and may complicate and result from a laceration of the brain. In such cases the inflammation generally commences within two or three days of the injury, and, spreading over the cerebral cortex from the seat of laceration, causes a diffuse suppuration beneath the pia mater, the meningitis being caused by an extension of inflammation from the subjacent grey matter. In my experience this form of intra-cranial suppuration is very rare.

Second, in cases of severe contusion the bone forming the vault of the skull may become acutely inflamed, just as the tibia might from a similar injury. The acute osteitis is liable to terminate in necrosis of the bone and in the formation of pus around it, and although this necrosis is in some cases limited to the external table, in others the inner table also necroses, and then the pus accumulates between the dura mater and the bone as well as beneath the scalp. The collection of pus beneath the scalp gives rise to a swelling of an inflammatory nature, to which the name of "Pott's puffy swelling" has for many years been applied, and which differs in no way from similar swellings formed in connection with diseased bone elsewhere. If the swelling be incised, bare dead bone is found beneath it, and, as in the case of acute osteitis elsewhere, pyæmia is a common complication. On account of the collection of pus between the

dura mater and the bone symptoms of compression of the brain may ensue, and if no treatment be adopted death may result from this cause, or else from an extension of inflammation to the arachnoid cavity and the supervention of diffuse meningitis. Symptoms of this form of intra-cranial suppuration do not usually supervene till about eight or ten days after injury.

Third. Abscesses may form in the brain itself, and probably result from laceration of the cerebral substance. Cerebral abscesses resulting from injury are usually of very slow formation, and may attain a considerable size in the substance of the hemispheres without producing any definite symptoms until they reach the grey matter of the cortex or of the large ganglia at the base. It may thus be weeks or even months after an accident when the first symptoms of cerebral abscess supervene.

Fourth. In cases of fracture of the cranial bones meningitis going on to suppuration may result either from irritation of the membranes by fragments of the inner table, or from the extension of sceptic inflammation from the outside in cases of compound fractures. This form of intra-cranial suppuration may occur at almost any period after the infliction of the injury.

HERNIA CEREBRI.

Hernia cerebri is a protrusion of brain-substance through an aperture in the cranial bones resulting from injury. It usually complicates compound fractures of the vertex in which the dura mater has been torn, and is the consequence of inflammation of the exposed and oftentimes injured cerebrum.

The protruded mass, or hernia, in these cases consists of brain matter and of granulation tissue, and contains more of the latter than of the former. It varies in size, but is seldom larger than a hen's egg; its colour is bright red; it is soft and vascular, and pulsates synchronously with the heart. The aperture in the bones through which it passes

is commonly much smaller than is the most prominent part of the swelling.

The course of cases of hernia cerebri differs greatly. When the subjacent brain is inflamed death commonly ensues, but in many cases where the exposed brain is kept clean and aseptic the meninges around the hernia become adherent, the latter soon ceases to increase, cicatrisation of the skin wound ensues, and as the scar tissue forms and contracts, the hernia itself gradually shrinks, and is finally covered in by fibrous tissue. I have several times seen cases run this favourable course.

CHAPTER LXVI.

SURGICAL COMPLICATIONS OF MIDDLE EAR DISEASE.

Inflammation of the middle ear or tympanic cavity may be caused in almost innumerable ways, and, although sometimes very acute in its onset, more often runs a subacute course. It is very commonly a sequel of scarlatina and measles, and in other cases is caused by exposure to cold. It is very frequently due to extension of inflammation from the pharynx, tonsils or naso-pharynx, and in its more chronic forms is commonly the result of some obstruction to nasal respiration, such as adenoid growths. The inflammation is, at first, almost invariably catarrhal in nature, but when untreated it is very liable to go on to suppuration, and when this occurs the membrana tympani is usually perforated, or in part destroyed in the extension of the suppuration. In many cases the perforation in the membrane heals and the hearing is recovered; but, especially when no treatment is adopted, the perforation may not close, and a chronic muco-purulent catarrh may persist for years. It is especially in cases of this class that serious intra-cranial complications may ensue, for, when the tympanic cavity has once become the seat of septic infection, further changes may result, which prepare the way for more serious affections.

When a chronic muco-purulent catarrh has become established the inflammation is liable to cause ulceration of the mucous membrane and extension of the inflammatory process to the ossicles and to the bony walls of the tympanic

cavity, conditions which often result in necrosis of the ossicles and caries of the temporal bone. In this manner the tympanic cavity may be enlarged by destruction of its bone, and the inflammatory process usually extends to the mastoid cells and affects in a similar way their lining membrane and bony walls.

Otitis media, apart from injury, is due to an infection by micro-organisms. The bacteriology of inflammation and suppuration of the middle ear has been carefully investigated in recent years. The organisms most frequently encountered in acute processes are the streptococcus pyogenes, the staphylococcus pyogenes albus and aureus, and the pneumococcus. These are occasionally found in pure culture; but more commonly several forms occur together. It is probable that these organisms wander into the tympanic cavity through the Eustachian tubes, and, the lining of this cavity being in a predisposed condition, produce inflammatory or suppurative changes; the same organisms have been obtained whether the otitis was suppurative or merely catarrhal or exudative. It is interesting to remark that pneumococci have been frequently found in cases of acute suppuration, for these organisms occur normally in the nose or mouth of many individuals. When otitis media occurs as a complication of acute infective fevers, as scarlatina, measles, diphtheria, influenza and small-pox, streptococci are most commonly found, although in a few rare cases of diphtheria the bacillus of Klebs-Löffler has been met with. In chronic otitis media, or in otorrhœa, the streptococci or pneumococci are found but seldom; generally staphylococci in combination with putrefactive organisms have been obtained from the fetid pus. In some cases of chronic ear disease the aspergillus niger, a black mushroom-like mould, has been seen, giving the pus and the tympanic cavity a peculiar dark stippled appearance. When the pus has a green or blue colour this is due to the bacillus pyocyaneus. The tubercle bacillus has also been found in the middle ear.

Cholesteatomata (margaritomata) are generally the result of chronic purulent otorrhœa, and are produced by an epidermoid change or metaplasia of the epithelium lining the tympanic cavity, by which this becomes squamous and horny. This cast-off epithelium is constantly shed into the pus, and is retained in the middle ear, where it dries up. Then, more and more scales are gradually added, and the dried mass becomes moulded into a round tumour, which fills up the middle ear, or the entrance of the mastoid cells, and acting as a foreign body begins the chronic inflammatory process and thereby favours its own growth. In appearance these cholesteatomatous masses are white and glistening in colour, and on section consist of concentric layers of large squamous cells, amongst which pus, débris and cholesterine crystals are found. They vary in size from a millet seed to a pigeon's egg. Not all cholesteatomata, however, are due to a retention of shed squamous epithelium in diseased ears, for some seem to be due to a primary growth in the mucous lining of the middle ear or in the dura-mater. Virchow was the first to describe these growths, and his description applied to glistening bodies which he detected in the dura-mater. It is a matter of some difficulty to explain their origin there, and the subject requires re-investigation. We may, however, assume quite safely that the cholesteatomata which are found in connection with chronic middle ear disease are not actual new growths, but retention products, epithelial sequestra.

The **complications** of middle-ear disease are many and serious, and may be classified as follows:

(*a*) **Acute infective osteitis.**
(*b*) **Sinus phlebitis.**
(*c*) **Meningitis.**
(*d*) **Cerebral and cerebellar abscesses.**

(*a*) **Acute infective osteitis.**—It has already been explained that after prolonged middle-ear disease there is often some caries and chronic osteitis, and it is easy to see that bone in such a condition may very easily become

more acutely inflamed at any time from exposure to cold, the retention of inflammatory products, or the admission of irritating matters or fluids into the middle ear. The results of this acute osteitis are most evident in the mastoid region, and the pus commonly makes its way to the surface of that bone behind the ear, and causes the formation of a mastoid abscess. In other cases, however, which are untreated, the periosteum may be stripped off the cranial bones, and the pus may burrow widely beneath the scalp, causing a diffuse cellulitis of this region which may completely mask the original ear trouble. More rarely the matter extends downwards to the under-surface of the mastoid bone, and passing beneath the attachments of the muscles in this situation, may burrow into the tissues of the neck. But serious as these complications are, the case becomes still more serious when, as the result of the osteitis, the pus makes its way to the cranial aspect of the bone, and forms a "sub-dural abscess" between the dura-mater and the skull; it very rarely happens that recovery can result in such a case without surgical assistance, and the inflammation is peculiarly liable to extend and cause "sinus phlebitis" or diffuse meningitis.

In all cases of acute osteitis the condition of the patient is much the same as it is when one of the long bones is attacked by the same disease, and high fever, and, perhaps, rigors and symptoms of pyæmia are to be expected. (*Vide* "Diffuse Periosteitis," &c., p. 310.)

(*b*) **Sinus phlebitis** is the term commonly employed to indicate inflammation of the cerebral sinuses, and especially of the lateral sinus. This complication is almost always the result of a direct extension of inflammation from the mastoid bone to the dura-mater enclosing the sinus, and its development is quickly complicated by the formation of clot on the inner surface of the inflamed wall. In most cases, unfortunately, the mischief does not end here, for, the inflammatory process being an infective one, micro-organisms extend into the coagulum, and

bacterial poisons find an entrance into the venous stream. The coagulation of the blood often extends a little way down the internal jugular vein, and may even reach as far as the left innominate vein. In most cases the passage of blood through the sinus is arrested by the clotting, but in some a channel is kept open by the flowing blood, which thus passes to the heart over a surface composed of disintegrating septic clot, portions of which are constantly washed into the general circulation.

In cases of sinus phlebitis the symptoms are essentially those of pyæmia, rigors being frequent and the temperature very high. When the jugular vein is involved in the neck there may be great tenderness and difficulty of movement, but the same symptoms may also be due to inflammation of the lymphatic glands in this situation, resulting from the neighbouring infective process.

(*c*) **Meningitis**.—This may occur either as a circumscribed serous effusion, or as a diffuse purulent inflammation of the pia-mater and arachnoid, the latter being unfortunately the more common. Meningitis may result either from the extension of suppuration from a sub-dural or a cerebral abscess, or else from a direct extension of inflammation from the inflamed bone, more especially in cases where the roof of the tympanic cavity is involved. The symptoms may be either those of meningitis alone or of the other affections to which the meningitis itself owns its cause.

(*d*) **Cerebral and cerebellar abscess**. — Abscess in any part of the brain is usually the result of extension of an infective inflammation from the bone to the cerebral substance along the course of a small vein or a lymphatic. It does not commonly complicate either acute osteitis, sub-dural abscess, or sinus phlebitis. The usual positions for such abscesses are the temporo-sphenoidal lobe in its anterior part—resulting from a spread of inflammation through the thin bone forming the roof of the tympanum—and the lateral lobe of the cerebellum ; the latter position is much the more uncommon.

The symptoms in cerebral abscess are referable to the affection of the brain substance, and are not usually those of blood poisoning from absorption of the products of inflammation, the temperature being usually slightly raised or even sub-normal, and the suppuration running a very slow course. So long as the pus is confined within the white matter of the temporo-sphenoidal lobe, and the abscess does not by its size cause pressure on the cortex, the symptoms may be limited to marked slowness of pulse, occasional vomiting, slow cerebration, drowsiness and slow speech, with, perhaps, optic neuritis; but, when the cortex is involved, muscular spasms, epileptiform convulsions commencing in one limb or in the face, and sometimes complete paralysis of one limb, and even hemiplegia may occur.

CHAPTER LXVII.

SPINA BIFIDA AND CONGENITAL SACRAL TUMOURS.

A SPINA BIFIDA is a congenital deformity of the spinal canal, with protrusion of a portion of its contents in the form of a cystic tumour.*

The bony deformity which is present in these cases consists of a deficiency in the laminæ and spines of one or more vertebræ, the spinal canal being thus left unclosed posteriorly. The laminæ may be merely stunted, but in bad cases are completely everted so as to stand out at right angles from the axis of the spinal column. As rare variations may be mentioned—(*a*) bony outgrowths from the posterior surface of the bodies of the vertebræ into the spinal canal, which may protrude through the centre of the cord itself; (*b*) cleavage of the body of a vertebra, and protrusion of the membranes anteriorly.

FIG. 175.

Portion of a Spine from a case of Cervical Spina Bifida, showing the absence of the laminæ and spinous processes.

The common **position** of a spina bifida is the lumbo-sacral region, but it may occur in any part of the spine. In most cases some five or six of the vertebræ are deformed,

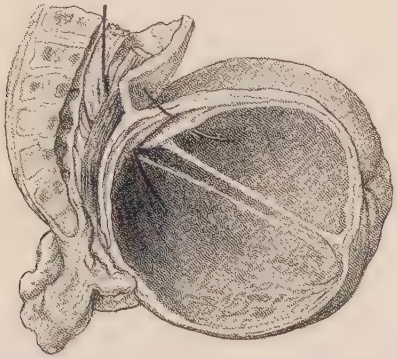
* For a full account of the morbid appearances found in different cases of spina bifida, see a report of a committee of the Clinical Society in vol. xviii. of their "Reports," from which this chapter has been in great part compiled.

but in exceptional instances the whole of the laminæ are deficient from the atlas downwards. Between these conditions all grades may be seen.

The **structure of the sac** and the composition of its contents differ in different specimens, and allow of a subdivision into three classes — first, meningocele; second, meningo-myelocele; third, syringo-myelocele.

In **Meningocele** the sac is composed of dura mater and arachnoid, but contains neither the spinal cord nor any nerves, and is distended by the sub-arachnoid or cerebro-

FIG. 176.



Spina Bifida.—A Meningo-myelocele. One bristle has been passed from the sub-arachnoid space into the sac, and another has been placed beneath the spinal cord just before the latter becomes spread out on the upper part of the sac-wall.

spinal fluid. It is doubtful whether one layer of the arachnoid alone is ever protruded, though this condition has been described. In such cases the sac would contain the arachnoid fluid.

In **Meningo-myelocele,** which is the most common form of spina bifida, the sac is formed by the dura mater and arachnoid, and contains cerebro-spinal fluid, with the spinal cord and its nerves. In lumbo-sacral spina bifida the **spinal cord** usually passes directly backwards, and the filum terminale is found adherent to the lower and posterior

portion of the sac. In some specimens the cord becomes adherent to the upper part of the sac as soon as it enters it, and is spread out over, and incorporated with, the sac-wall itself. In cases of spina bifida in the dorsal or cervical regions the spinal cord passes through the sac, re-enters the spinal canal, and is found in its natural position in the lumbar region. In its passage through the sac it may be free, but is in some instances adherent to the posterior surface of the sac-wall, as in the lumbo-sacral class. In all cases of spina bifida the central canal of the cord is liable to be distended.

The **nerves** which arise from that part of the spinal cord which is included in the spina bifida are necessarily themselves within the cavity of the sac, the anterior and posterior roots being often easily differentiated and found separated from one another by a continuation of the ligamentum denticulatum. That part of the cord which is fused with the posterior portion of the sac-wall also gives off nerve-roots, and thus the latter appear to *originate* from the sac-wall itself, though they are often erroneously described as being *distributed* to it. (See Fig. 176.)

The **form** and **size** of the **sac** vary greatly. At birth it is usually small, not larger than a Tangerine orange, whilst in some cases the position of the spina bifida is marked by a depression instead of a swelling. During the first few days of life the sac increases with some rapidity, and has been seen to measure in adults as much as 27 inches round its base. Its shape is circular, and, although generally sessile, there is a marked constriction where it is connected with the trunk; in long-standing cases a pedicle is gradually formed. The most posterior portion of the sac, or the summit, frequently presents a medium groove, furrow, or depression, to which the name of "the umbilicus" has been given. It is generally the result of the attachment of the spinal cord in this situation, and is caused by the unequal distension of the sac by its contained fluid. (See Fig. 177.) The central canal of the spinal cord has

been seen to open at the bottom of the umbilicus. Occasionally, the sac is divided into partitions by septa, and these also may produce depressions or grooves.

The **coverings of the sac** are rarely normal skin and subcutaneous tissue. In most cases the base alone is covered by skin, which gradually thins away towards the fundus, so that the summit of the latter is either covered by a thin layer of epidermis or by a glistening membrane composed of the spinal membranes or of the thinned and adherent cord.

Syringo-myelocele is a rare form of spina bifida in

FIG. 177.

Spina Bifida, showing the umbilicus or central depression, where the spinal cord is adherent to the sac-wall.

which the central canal of the cord is immensely distended and forms the sac-cavity, the sac-wall being lined by the expanded spinal cord itself. At first sight such a spina bifida might easily be mistaken for a simple meningocele, for no nerves traverse the sac-cavity. Close examination, however, shows that the nerves are contained in the substance of the sac-wall, being given off from the distended cord.

The coverings of a syringo-myelocele are of the same nature as those of a meningo-myelocele.

The congenital deficiency which results in the formation of any spina bifida consists in an imperfect development

of the mesoblast on each side of the vertebral furrow from which the spinal meninges and the vertebral laminæ are normally produced. The mesoblast being deficient, these structures remain undeveloped in varying degrees.

The complications of spina bifida which are common are hydrocephalus, talipes, and paralysis of the rectum and bladder.

The termination of the majority of cases of spina bifida is death. This commonly results from the sac giving way, an event which is followed by myelitis or convulsions. Other children die in a marasmic condition, without any very definite lesions being present. In a small proportion of the meningo-myeloceles, and in a considerable number of the simple meningoceles. the sac gradually shrinks and a natural cure is effected.

CONGENITAL SACRAL TUMOURS.

The congenital tumours which occur in the sacral and coccygeal regions may conveniently be divided into three classes—(1) dermoid cysts and included fœtuses; (2) sacral cysts and sacral lipomata; (3) coccygeal cysts.

1. **Dermoid cysts** are commonly met with in the middle line of the back and are covered by normal skin. In many cases the tumour is an irregularly globular mass of varying consistency, and on section is found to contain sebaceous matter and hair, with which there may also be found cartilage, teeth, and bone. These cysts may have deep attachments, and may be connected with the spinal membranes.

In another class of cases there is a definite projecting appendix protruding from the exterior wall of the cyst, and bearing a more or less close resemblance to a tail. In other examples, again, the protruding portion terminates in ill-developed fingers or toes, and on dissection is found to be an imperfectly formed limb. Progressing onward from this, transition is easy to a fully developed lower extremity, which, however, is rarely as large as the normal

limbs; the name of "human tripod" has been applied to infants with such an appendage. Further stages of development of the attached fœtus may also be found. Thus, instead of a single projecting limb there may be two lower extremities, and a double pelvis; or there may be in addition the trunk and extremities of a so-called "parasitic fœtus;" whilst, finally, two well-formed fœtuses may exist attached to each other in the sacral region, and forming one of the varieties of "double monster."

It will thus be seen that between a simple dermoid cyst in the sacral region and a fully developed fœtus all stages may exist, but it should be remembered that in any case the attached fœtus or the cyst may have close connection with the spinal column or the meninges.

Such tumours as these result either from simple inclusion of epithelium in the closure of the vertebral groove, as in the case of dermoid cysts elsewhere, or else from the development of two embryonic areas in a single blastodermic vesicle, and all cases of attached or included fœtus or parasitic fœtus are to be explained by the latter hypothesis. It is known that the rudiment of the embryo appears primarily as an opaque spot on the blastodermic vesicle, which spot is known as the embryonic area. If two of these embryonic areas form on a single vesicle and subsequently in their development coalesce, two joined fœtuses are formed, and the union may be either in the sacral region, or in other parts such as the thoracic or abdominal walls or the crania. In most cases one of the two fœtuses is never perfectly developed, and remains as a shapeless mass of bone, cartilage, &c.; in other cases the development of the two fœtuses is more equal. It is probable that in the cases of imperfect development the imperfection is the result of deficient blood-supply or of pressure by the other fœtus. It is, however, also possible that some cases of imperfectly formed double limbs are the result of duplication of parts which are naturally single, and that in these cases there was never more than a single

embryonic area, but rather a development of an extra limb from cells which should normally be differentiated to form merely a pair.

2. **Sacral cysts** may also develop independently of inclusion of epiblast or ova, and appear to be of mesoblastic origin. They probably are formed, as are the cystic hygromata of the neck, from portions of mesoblast, which are so to say in excess, and which remain when the requisite muscular and osseous structures have been developed. They consist of a soft fibrous wall lined by endothelium,

FIG. 178.

A Unilateral Coccygeal Cyst.

and containing serous fluid. In addition to these, fatty tumours, or "caudal lipomata," are rarely found, and are analogous to the deeply seated congenital lipomata already described on page 166, as being seen occasionally attached to the bones of the extremities. Both the cysts and the lipomata may have attachments to the vertebræ, or even to the membranes, but the latter is not nearly so common an occurrence as in the case of the dermoid cysts. Both of

these forms of congenital tumour are usually found a little to one side of the middle line.

3. **Coccygeal cysts** are placed lower down than the sacral tumours just described. They form tense fluid swellings of considerable size, are always placed laterally, and occupy chiefly the gluteal region, by the muscles of which they are covered. Dissection shows that these tumours consist of a dense fibrous wall of considerable thickness at its attached margin, but often very thin at its most projecting part. This wall is especially fixed to the lateral and anterior aspects of the coccyx and the lower part of the sacrum, where there is generally a considerable amount of solid growth. The cyst commonly extends into the pelvis, and pushes forward the rectum and anus, whilst laterally it protrudes between the coccyx and the pelvis into the gluteal region, and bulges the skin below the margin of the gluteus maximus, and tends to protrude between the thighs.

The tumour lies beneath the levator ani muscle, and in consequence of its pressure the coccyx is commonly straightened, or its extremity may even point backwards.

The amount of the cyst within the pelvis differs much in different cases. At present I have a child under observation in whom it extends above the promontory of the sacrum, and have examined dissected specimens in which the growth extended as far as the promontory.

The cysts contain a very viscid blood-stained fluid, as well as a certain amount of solid ingrowth. This, in a case I have recently dissected, looked very much like a mass of large nasal polypi, as it consisted of soft, friable, red tissue, hanging in a pendulous manner from that part of the cyst which was attached to the coccyx and attached to its wall in numerous places by slender pedicles.

In another specimen, shown by Mr. Shattock at a meeting of the Pathological Society, there were multiple cysts, and a mixture of cartilage, bone, and fibrous tissue in the stroma.

In several of the recorded cases gland-tissue resembling that of the thyroid has been found, either forming the bulk of the growth, or else occurring in connection with cartilage and fibrous tissue.

The mode of origin of these coccygeal cysts is still a matter of doubt, though on account of their position and of the gland-tissue which they contain, it is probable that they originate, as has been suggested by Middledorpf and Sutton, in the post-anal gut.

CHAPTER LXVIII.

TALIPES—CLUB-FOOT.

Talipes, or club-foot, may be either congenital or acquired, may affect one or both feet, and is equally common in male and female children.

CAUSES.

The **congenital** form is attributed by different authors to three different causes—(1) spasmodic contraction or paralysis of muscles, resulting from nerve-irritation or loss of function; (2) malformation of the tarsal bones; (3) mechanical pressure by the uterus, or by malposition of the fœtus.

The theory that the deformity is due to some affection of the nervous centres is supported, first, by the undoubted fact that after birth talipes certainly results in some cases from disease of the spinal cord, and, second, by the very frequent association of malformations of the cord and brain such as are seen in spina bifida and anencephalus.

In the absence of any demonstrations of primary disease of the spinal cord in the fœtus, the proof that myelitis produces congenital talipes must be declared wanting, although there is much to be said in its favour from a theoretical point of view. The frequent association with spina bifida and anencephalus is, however, a fact which renders it highly probable that their co-existence is more than a coincidence, and that, in these cases at any rate, the deformity of the foot is the direct result of the lesion of the cord which is commonly present.

The second theory, which explains the deformity by imperfect development of the tarsal bones, is probably erroneous, the changes found in the latter being the result, and not the cause, of the talipes.

The third theory, that the club-foot is the result of mechanical pressure by the uterus or by adventitious bands, or of malposition of the fœtus, finds its greatest support in a paper by Messrs. Parker and Shattock,* and may be summed up in their own words :—"Our argument is that the feet of the fœtus occupy various positions during the course of intra-uterine life, and that this occurs in order that the joint-surfaces, the muscles, and especially the ligaments be developed so as to allow of that variety of positions and movements which are afterwards to be natural to the foot ; and we hold that when anything (mechanically) prevents the feet from assuming these positions at their proper time, or maintains them in any given position beyond the limit of time during which they should occupy such position, a talipes result. The variety of talipes will depend on the date of its production ; its severity will be in direct ratio to the mechanical violence at work. If the inversion of the foot, which is normal during the earlier months of fœtal life, be maintained beyond the normal period of time, the muscles and ligaments will as a consequence be adaptively short on one aspect of the limb, and too long on the other—a normal position of inversion will finally become a deformity. Talipes calcaneus is, we believe, produced in a similar manner ; it occurs, however, later during intra-uterine life, when a flexed position of the foot is normal. Being thus less fundamental in character, as a deformity it is also less severe than varus."

In support of this explanation the authors adduce various examples of fœtal malposition and pressure which seem quite sufficient to prove that the theories they advocate are supported by facts, and to establish such mechanical condi-

* "Transactions of the Pathological Society," vol. xxxv. p. 423.

tions as one of the causes of congenital club-foot, and probably the one which is most frequently operative.

Acquired talipes commonly results from disease of the spinal cord. This in some cases is irritative in its nature and produces spasm of certain muscles, which, at first by their vital contraction, and afterwards by their tendency to became permanently shortened, cause an active displacement of the foot—*e.g.*, spasm of the calf-muscles will result in a drawing up of the heel, or "talipes equinus."

In another class of cases the lesion of the cord is of a destructive nature, the large nerve-cells in the anterior cornua being destroyed. This lesion, which is known as "anterior polio-myelitis," results in the condition called "infantile paralysis." The motor cells, which are the centres of nutrition for the motor nerves and the muscles they supply, being destroyed, the nervous and muscular fibres degenerate and atrophy, and deformities are produced both by shrinking of the paralysed muscles as well as in other cases by their stretching. Thus, if all the muscles of the leg are paralysed, the foot usually hangs in a helpless condition, with the toes pointed and somewhat inverted, and in this position, that of "talipes equino-varus," it may be permanently fixed in the course of months by the atrophy and shrinking of the calf-muscles. In other cases, where the calf-muscles are alone or chiefly affected, the extensors will support the foot, and the pressure on the sole in walking will tend to stretch the wasted soleus and gastrocnemius until the heel is permanently depressed and "talipes calcaneus" is produced. In some cases of polio-myelitis the paralysis passes off, but unless this improvement occurs within the first month or two after the attack there is little probability of recovery; the muscles then rapidly waste, the skin becomes blue and cold, and, unless precautionary measures are taken, deformities such as have been described will be developed. In advanced cases the muscles consist of little else than fat.

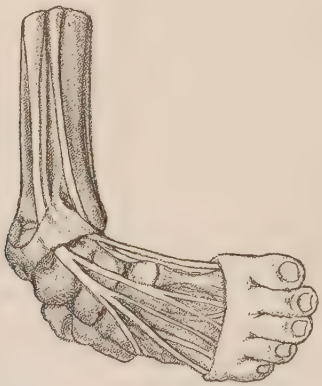
In a few cases talipes is caused contraction of scars

about the ankle-joint, and, as will be explained in the sections on flat-foot and valgus, it is also the result of muscular weakness and yielding of ligaments. Again, when one limb is shorter than the other—*e.g.*, as the result of hip disease—the constant walking on the toes may produce permanent talipes equinus.

TALIPES EQUINO-VARUS.

Talipes equino-varus is the commonest form of club-foot, and is in most cases congenital. In this variety the toes are pointed and the sole cannot be brought up to a right

FIG. 179.



Dissection of a Foot with Talipes Equino-varus.

angle with the leg; the foot is also turned inwards so that the scaphoid bone is approximated to the internal malleolus. In severe cases the patient walks on the outer side and dorsum of the foot, and, as a consequence, the skin in this situation becomes much thickened, and a subcutaneous bursa usually forms. If such a deformed foot be examined, the tendons of the tibialis anticus and the tibialis posticus as well as the tendo-Achillis are found to be abnormally short; the internal lateral ligament of the ankle and the ligaments uniting the tarsal bones on the inner side are in

a similar condition, whilst those on the outer side may be stretched. The astragalus is in most cases normally placed as regards the tibia and fibula, except that, in proportion to the amount of the equinus, it is depressed anteriorly so that the greater part of the surface which articulates with the tibia is exposed. The twisting inwards of the foot, or the "varus," is found to take place at the mid-tarsal and sub-astragaloid joints, and not at the ankle, the scaphoid being displaced almost completely in severe cases to the inner side of the astragalus, so that the articular surface of the head of the latter bone is unopposed in front, and a new articular facet is formed on the inner side of the head to articulate with the navicular bone in its altered position. In addition to this, Messrs. Parker and Shattock have noticed an extension backwards of the upper articular or trochlear surface, and an undue obliquity of the neck of the astragalus.

In old cases of equino-varus, moreover, the bones on the inner side of the foot are compressed and undeveloped, the skin on the outer side forms a hard, horny excrescence, and the muscles of the calf are small and feeble from the little use to which they are put, the movements of the ankle-joint being in these patients almost in abeyance.

TALIPES EQUINUS.

Talipes equinus consists in an inability to bring the sole of the foot up to a right angle with the leg. The toes are consequently pointed, and the heel is raised from the ground in walking. In exaggerated cases the plantar flexion of the foot is carried to such an extent that the patient walks on the dorsal surface of the toes.

Pure equinus is rare. It is seldom congenital, and commonly results from infantile paralysis or from spastic contraction of the calf-muscles. The chief anatomical lesion is shortening of the tendo-Achillis or of the muscles inserted into it.

TALIPES VARUS.

Talipes varus uncomplicated with equinus is a rare form of club-foot, and is almost always congenital. It is characterised by inversion of the foot and shortening of the tibialis anticus and posticus. Most of what has been written already on the anatomical changes in talipes equino-varus applies also to varus.

TALIPES CAVUS.

Talipes cavus is an exaggeration of the normal plantar arch, the sole of the foot being deeply hollowed and the dorsum correspondingly convex and arched. This form of club-foot is invariably acquired, and is almost always combined with equinus or equino-varus. The toes also, in this variety of deformity, present conditions which are seldom seen apart from cavus, the first phalanges being hyper-extended or even dislocated backwards on their metatarsal bones, whilst the second and third phalanges are doubled down towards the sole.

This "hollow claw-foot" results either from a paralysis of the interossei, or in consequence of an antecedent equinus. The interossei normally flex the first phalanges and extend the second and third; consequently, when they are paralysed, there being no muscle to depress the first phalanges during the movements of the foot, the common extensor displaces them backwards onto the heads of the metacarpal bones.

In cases of equinus, again, the dislocation backwards of the first phalanges is produced by the efforts of the common extensor (together with other muscles) to maintain the foot in its natural position. Further, as regards the cavus, when the patient constantly walks on his toes, there is not only a tendency for the latter to be displaced by the weight they support, but the arch of the sole is increased by the contraction of those muscles whose tendons pass across it, especially the tibialis posticus and the peroneus longus. In

all cases of long standing, the plantar fascia is thickened and contracted, and offers material resistance to attempts at replacement.

TALIPES PLANUS.

Talipes planus, or flat-foot, is an acquired deformity, and results from muscular overwork and consequent weakness and loss of tone. The arch of the foot does not exist in an infant, and is only developed when the child begins to walk, being, in fact, produced by the contractions of the muscles whose tendons pass across the sole. The arch thus formed is also maintained by the use of the muscles, and the weight of the body does not rest, or depend for its support, directly upon the ligaments. If, therefore, the muscles, from overwork, lose tone and become weak and flabby, the weight of the body is no longer maintained by their healthy contraction, the foot is not braced by their support, and the ligaments, now called upon to do more than they were originally intended for, yield and stretch beneath their burden.

All the ligaments of the sole may be more or less implicated, but those which are most notably stretched are the inferior calcaneo-scaphoid and the long plantar. The head of the astragalus, being no longer supported, sinks down on to the soft structures forming the sole, and the bone, at the same time sliding forwards, pushes the scaphoid in front of it, and causes a displacement of the anterior half of the tarsus to the outer side. In the early stage of flat-foot, the head of the astragalus can be replaced, and the arch temporarily restored by artificial pressure; but in cases of longer standing this is not possible, and the whole foot becomes rigid and stiff. The pressure of the displaced astragalus also gives rise to much aching pain, which is increased by walking. The deformity is most common in growing boys and girls about the age of puberty who are employed in some occupation which necessitates prolonged standing.

Another cause of flat-foot deserves mention, although briefly. It is liable to supervene in patients who suffer from chronic rheumatism, and it sometimes occurs in those who suffer from so-called gonorrhœal rheumatism.

TALIPES VALGUS.

Talipes valgus consists of a displacement outwards of the tarsus at the mid-tarsal and sub-astragaloid joints, and is practically an exaggerated condition of talipes planus. It may develop out of an ordinary flat-foot, or result from infantile paralysis of the muscles of the calf and inner side of the leg in patients in whom the peronei and extensors escape. In such cases it is sometimes complicated by calcaneus. In talipes valgus the arch is flattened, the foot is everted, the cuboid bone is approximated to the external malleolus, and the head of the astragalus forms a projection on the inner side. The peronei muscles are contracted, and resist efforts at replacement of the foot.

TALIPES CALCANEUS.

Talipes calcaneus is a rare form of club-foot. In it the heel is depressed and the toes are drawn up towards the

FIG. 180.

A Foot with Talipes Calcaneus.

shin. It is in some cases congenital, but in others results, as already explained, from the stretching of the atrophied calf-muscles in cases where the latter are affected by infantile paralysis. The chief anatomical abnormality is undue length of the tendo-Achillis or of the muscles inserted into it, with consequent inability to stand on the toes or to propel the body forwards in the act of walking. It is combined in some cases with valgus.

CHAPTER LXIX.

GENU VALGUM.

Genu valgum, or knock-knee, is an outward displacement of the tibia on the femur, with consequent separation of the internal malleoli and the feet, and a tendency for the knees to rub against one another in the act of walking.

Causes.—Unlike most other deformities, genu valgum is never congenital. It is developed in young children in consequence of rickets, or else at puberty—and seldom later—from muscular weakness and overwork.

In rickety children the femur tends to become bent in an outward and forward curve in the upper and middle parts of its shaft, with the result that there is developed, first, a tendency for the axis of the legs to cross, and, secondly, a compensating curve of the lower third of the bone, with the convexity inwards. But although this secondary curve is, in a limited degree, of use, nevertheless, by directing the lower articular surface of the femur outwards, it causes the knee-joint to be placed obliquely instead of transversely across the long axis of the lower extremity, so that the weight of the body is transmitted down a line which falls through the outer condyle to the inner side of the foot. If the rachitic and softened state of the bones continues the curve tends to increase, and the tibia is directed further and further away from the middle line, whilst, in addition, the knee-joint being no longer placed transversely to the axis of the limb, the internal lateral ligament is placed at a disadvantage, stretches, and allows

the tibia to be displaced from its articulation with the femur.

In adults, knock-knee is primarily due to muscular weakness, the knee-joint, like the sole of the foot, depending for its integrity, not only on ligaments, but also on muscles. If, therefore, from overwork in young and growing patients, the muscles lose tone, the ligaments are soon unable to maintain the articular surfaces in their normal position and genu valgum results. The reasons why the

FIG. 181.

Rachitic Knock-knee.

tibia is displaced outwards, and the internal lateral ligament yields rather than the external, are that, the foot and leg being normally slightly rotated outwards, the strain is naturally thrown on the inner side, and that the fascia on the outer side of the joint is much stronger and denser than that on the inner, and therefore less likely to yield and stretch.

From whatever cause arising, the deformity tends to increase. The whole weight of the body is now placed on the external condyle and outer articular surface of the

tibia, the internal condyle and inner articular surface being separated slightly from one another. As a consequence, the growth of the external condyle is arrested by the constant pressure, whilst that of the internal continues, and the necessary result is a relative elongation of the internal condyle, which then acts as a mechanical obstruction to all attempts at replacement of the displaced tibia. The soft tissues, also, on the outer aspect of the limb accommodate themselves to the altered position of the parts, and in time become contracted and shortened to such an extent as to resist all attempts at forcible straightening; the structures in such cases which are most unyielding are the fascia lata, the biceps, and the external lateral ligament.

In **genu varum** the knee is bowed outwards instead of inwards, so that, whilst the feet tend to knock against one another, there is a considerable space between the knees. Genu varum commonly results from rickets, and is much more rare than genu valgum.

CHAPTER LXX.

CONGENITAL DISLOCATION OF THE HIP.

Congenital dislocation of the hip is met with much more frequently in female than in male children, and may occur

Fig. 182.

Congenital Displacement of the Right Femur, showing the head of the bone included within the capsular ligament. (From the museum of St. Thomas's Hospital.)

on one or both sides. It occasionally affects several members of the same family, and may be hereditary.

The head of the femur is displaced upwards and back-

wards, and the shaft is rotated inwards. The capsule is exceedingly loose, and allows of very considerable movement of the femur on the ilium. The head of the femur is always more or less flattened by pressure, and in some few cases has been found undeveloped from birth. The dorsum ilii is flattened or slightly hollowed by the constant pressure of the femoral head. The gluteal muscles are

FIG. 183.

Congenital Displacement of the Femur, showing the altered acetabulum, the roughened spot where the head of the bone has rested, and the flattened femoral head. (From the museum of St. Thomas's Hospital.)

usually fatty and ill-developed, on account of the little use to which they are put in the altered position of the parts.

The condition of the acetabulum is, however, the most important feature, for in all the specimens in the museums of London it is very imperfectly developed. The developmental deficiency is also remarkably uniform, and consists in a suppression of that part of the cavity which is formed by the ilium—of what may be called the iliac segment. The acetabulum, in consequence, is formed by the pubes

and ischium alone, and, instead of being round, is of a triangular shape, with the apex upwards. It is, in addition, very shallow, with margins scarcely raised above the level of the surrounding bone.

It is now tolerably certain that the failure of development of the acetabulum is the real cause of the displacement of the femur, and there is little doubt that the theory which has been accepted recently, that the dislocation is a traumatic one produced by the accoucheur during delivery, is erroneous. In most cases there is no history of any difficulty at birth; whilst the facts that the displacement is occasionally hereditary, that it affects chiefly female children, that it is often double, that the head of the bone is always within its capsule, and that no case has yet been described in which any laceration of the tissues has been found, render it highly improbable that difficulties during parturition have anything to do with the causation of the deformity.

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